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MASSACHUSETTS MEDICAL SOCIETY

Section of Surgery

Presiding: Dr. Frederick S. Hopkins, Springfield, chairman; Dr. Augustus Thorndike, Jr., Boston, secretary.

THE CHAIRMAN: This symposium on traumatic surgery will be opened by a leading neurosurgeon who has added greatly to our knowledge of spinal injuries, Dr. W. Jason Mixter, of Boston, whose paper is entitled "Major Injuries to the Spine."

MAJOR INJURIES TO THE SPINE

W. JASON MIXTER, M.D.*

BOSTON

IN THESE days of rapid transportation and strenuous sport, major injuries to the spine are becoming commoner. The old industrial hazards are still with us, though happily the worker is more protected from them than he was years ago. A great deal has been written in the past few years on injuries of the spine, but these papers have been devoted largely to special fractures or to special phases of the subject.

As the time at my disposal is rather limited I shall omit all historical matter and reference to the literature and shall give my own opinions for what they are worth. I realize that I am dealing with a subject that is of importance to the general practitioner and also to various special groups, for these cases may and often do require the attention of those who are specializing in various branches of medicine.

Let us follow the course of a patient with a major spine injury, including the time he is hurt, his transportation to the hospital, the methods used to reach a diagnosis, and finally, though not in detail, the treatment to be used. We may see these patients first at the scene of the accident, at home, or in the receiving ward of a hospital, and our first actions may go far to determine the final result. Too often a serious complication can be traced to the first aid or the treatment given during the first few hours after injury.

Transportation of such a patient, whether paralyzed or not, should be planned so as to prevent damage to the spinal cord. If the injury is to the cervical or upper dorsal spine, he should be placed carefully on his back, on a firm but well-padded stretcher, and tied. An ironing board or a blind taken from the nearest house and covered with blankets is better than a stretcher that sags. The head must be supported at all times and not allowed to twist; above all, no flexion of the neck must be allowed. If someone can steady the head and exert gentle traction during transportation, so much the better; if not, it may be supported on both sides with blanket rolls or sandbags. If the fracture is dorsal or lumbar, the patient should be placed on his face with the head turned, so as to permit respiration.

On arrival at the hospital, the clothing should be cut away, and the skin dried and freed of gravel and dirt. A small stone, or even a suspender button, digging into the insensitive skin of a paralyzed patient, may be the starting-point of an intractable area of necrosis. The back should be examined for disalignment, kyphos, localized tenderness or swelling. Obliteration of the groove between the erector spinae muscle groups by edema is suggestive of a major spine injury. A test for gross loss of sensation should be carried out, and the patient told to move his legs. This should take only a few minutes, and is combined with a cursory examination for other injuries. Complete paralysis of all muscles and of all sensation, both superficial and deep usually, though not invariably, means complete and irreparable damage to the cord.

Presented at the annual meeting of the Massachusetts Medical Society, Boston, June 3, 1937.

*Visiting surgeon, Massachusetts General Hospital.

If the patient is in shock cover him with warm blankets and institute shock treatment. After shock, if any, has been treated and the patient is in a condition to warrant further investigation, it should be carried out. Even if there is no paralysis, a neurologic examination should be made, one of the most important points being the determination of the upper limit of loss of power or of sensory or reflex abnormality. The sensory level, if one is present, should be marked on the skin for comparison in future examinations. In order to carry out these procedures it is necessary to move the patient; and this can be done without undue risk if care is taken to move him without bending.

Next in order comes the x-ray examination. This should include both anteroposterior and lateral plates, the latter often being the far more important of the two. The roentgenologist should be told to focus over the suspected vertebra, and x-ray examination of the whole spine should never be ordered.

Time is valuable, and even if the injury occurs at night these steps should be taken at once.

At this point the question of a consultant arises. If there is evidence of cord or cauda equina injury, your choice may be a neurologist or a neurosurgeon; if there is no such evidence, he may be an orthopedist or someone interested in fracture work. But there is no hard and fast rule, as many experts on fractures are well versed in the handling of spinal injuries with involvement of cord or nerve roots. If a consultant is to be employed, he should be called at once, for, I repeat, time is valuable.

In cases of injury to the spine which show neurologic signs or paralysis, if such evidence persists more than an hour or two there is probable damage to the cord or cauda equina, and lumbar puncture is indicated. This should be performed as soon as practicable after the neurologic examination, and should not be postponed so as to see whether the patient will improve without it. Lumbar puncture in order to be of value must be performed at a point below the fracture; a manometer is essential. The two things to be determined are the presence or absence of hemorrhage, and the presence or absence of a block in the subarachnoid space at the point of injury. Compression of the jugular veins causes marked increase in intracranial pressure. If the canal is open this pressure is transmitted immediately to the lumbar sac and the spinal fluid shoots up in the manometer, falling rapidly to its original level as soon as the compression is released. If this occurs there is no block, and neither the cord nor the cauda equina is compressed. If the fluid does not rise, or rises and falls slowly, complete or partial block is indicated. This, in turn, is presumptive evidence of continuing compression of the contents of the spinal canal.

If the examiner has worked rapidly he has obtained, within a few hours of the injury, fairly complete knowledge of the case. He must now decide whether laminectomy to relieve cord compression is advisable.

The cardinal principle in injuries of the back with involvement of the spinal cord or cauda equina is that we must differentiate sharply which of these two structures is injured. The cord is a very vulnerable, very soft structure, the nerve fibers of which are not protected by sheaths. Their interruption is never followed by repair and regeneration. Compression with physiologic interruption may be followed by restoration of function only if relieved in a short time — by which we mean hours, not days, if the compression is due to injury. The cauda equina, on the other hand, is a much less vulnerable structure, and return of function to the nerve roots of which it is composed may take place even after a considerable period of compression. As a help in differentiating the two types of lesion it should be recalled that the cauda equina begins at the top of the first lumbar vertebra.

Cervical fractures with cord compression are very serious injuries. The cervical spine is of relatively weak construction, and laminectomy after fracture will weaken it still more. The cervical cord is a very important structure, and is closely associated with the heat-regulating and respiratory mechanisms. Laminectomy in cases of cervical fracture, for reasons which are not clear, seems to cause an increase in the disturbance of these functions, apparently making the mortality much higher than that from fracture alone. For this reason the best treatment is traction, with or without manipulation, even if block is present. Traction in order to be of value must be adequate, and it is often necessary to use that of thirty, forty or even fifty pounds; a few pounds' traction is not sufficient. On account of the force necessary, skeletal traction may be best borne by the patient if wires or tongs are attached to the skull. After good muscular relaxation has been secured, or after x-ray evidence that the deformity has been reduced is obtained, lumbar puncture is repeated. If the block has disappeared or is less than before traction was applied, laminectomy is not indicated. If it persists, laminectomy should be performed. Traction should be continued during and after the operation. I believe that laminectomy in the cervical region rarely is indicated; if performed, it should be done within twenty-four hours of the injury. After that time, it is rarely successful.

Fractures in the dorsal region with evidence of cord compression are treated quite differently from those in the cervical region, since there is little danger of dislocation after operation and the injury is farther away from the medulla. Laminec-

tomy should be performed on all cases where block is present, except in those rare instances where neurologic signs are diminishing. Here again the operation must be done soon after the injury. If the pressure on the cord is not relieved within twenty-four hours, the chances are that the damage is done and that operation will not aid or hasten recovery of function.

Fracture of the lumbar spine with injury to the cauda equina should be treated differently from cervical or dorsal injury. Laminectomy may be postponed until existing bony deformities have been corrected. If lumbar puncture still shows evidence of block and the signs of nerve damage do not diminish, operation is advisable.

Whether or not a laminectomy has been performed there is still the problem of reduction of the bony deformity. We have learned in the past few years that this is a matter of great importance. If laminectomy has shown that the damage to the cord is irreparable, reduction need not be attempted, but if there are no neurologic signs, or if restoration of function in the cord is hoped for, everything possible must be done to restore the normal contour of the spine.

The problem of fracture or dislocation of the spine with bony deformity is a difficult one, and should not be attempted without training in this field. The mere presence of a Rogers frame in a hospital is not enough; the surgeon must know how to use it. One never knows what may develop in these cases, and ill-advised manipulations may result in severe cord injury and disaster. Cervical fractures without cord injury are treated in much the same way as if the cord were injured, reduction of deformity being accomplished by traction and manipulation. The traction is kept up for several weeks and is followed by some well-fitting orthopedic apparatus. Immobilization in plaster immediately after reduction is perfectly good surgical practice. I mention this method because many of our leading surgeons prefer it, but I myself am in favor of traction. Fracture dislocation of the cervical spine has a definite tendency to recur. For this reason it may be advisable to fuse the spine or to lash the spinous processes or laminae together after reduction. This is sound surgery, and I believe that the method will come to be used more frequently than it has been.

Dorsal and lumbar fractures almost invariably show more or less collapse of the bodies of one or more vertebrae, and many authors have emphasized the need of reducing this deformity. This is just as important after laminectomy as in cases where no operation has been performed; great care must be used, however, not to damage the cord. Various methods have been devised; but I believe the most logical and safest is that of opening up the collapsed body with the patient on a Rogers frame, with immediate application of a plaster jacket in hyperextension. This is very successful in crush fractures below the mid-dorsal region, but less so above, as the deformity tends to recur. The upper dorsal region cannot be held as successfully in a jacket as can the lower part of the spine. Posterior dislocations should not be treated on a Rogers frame. Open reduction or manipulation may be advisable.

Much could be said about aftercare. The care of the bladder in patients with cord damage was ably presented here last year by Munro.* I wish only to draw your attention to his excellent presentation. Tidal drainage will save many lives. Bedsores and all the other complications which we may encounter, such as pneumonia and abdominal distention, must be guarded against from the very beginning. We do not like to move a patient with a broken back, and the patient will complain bitterly if this is done; yet to roll him on his side or even on his face and to keep him off his back as many hours out of the twenty-four as possible may prevent these complications. If plaster is used, even a shell, in a paralyzed patient, only the most extreme vigilance will prevent a bedsore, and often it will occur in spite of perfect nursing care.

I have outlined the early handling of cases of broken back, laying particular emphasis on cases with injury to the cord or cauda equina. I have discussed briefly the pros and cons of decompressive laminectomy. To make the story complete I should also discuss the operative procedures, both neurosurgical and orthopedic, that may be used. Time will not permit this, but I must mention one other type of injury, namely rupture of the intervertebral disk.

This lesion is probably responsible for a small percentage of the intractable sciaticas which result from low-back injuries. It does not cause pain in the leg unless rupture into the spinal canal occurs. Ruptures outside the canal or into the vertebral bodies are unimportant, and according to our present knowledge are not disabling. The symptoms of this lesion are so similar to those of sciatica and of sacroiliac disease or lumbosacral strain that it should not be considered as a possible diagnosis unless examination of the spinal fluid reveals a high total protein, or adequate and prolonged orthopedic treatment fails to bring relief. The diagnosis cannot be made except by the use of lipiodol and careful x-ray examination, followed by

*Munro, Donald: The cord bladder: its definition, treatment and prognosis when associated with spinal cord injuries. New Eng. J. Med. 215:766, 1936.

laminectomy and removal of the fragment. This lesion is an interesting one, and is important in that it is being diagnosed loosely and on insufficient evidence. Our knowledge of this subject is incomplete, and opinions that we hold at present may become untenable upon further study.

I close this discussion with a few bits of advice:

(1) The care of a broken back is important from the first minute one sees the patient.

(2) Always secure good x-rays of an injured back, even if the injury seems a minor one.

(3) Begin treatment to prevent a bedsore at once.

(4) A broken back with cord involvement is a real surgical emergency, and delay may be just as serious to the patient as it would be if he were suffering from a ruptured viscus.

319 Longwood Avenue.

Discussion

THE CHAIRMAN: The discussion will be opened by Dr. William Rogers, visiting orthopedic surgeon to the Massachusetts General Hospital.

Dr. Rogers then proceeded to demonstrate by means of lantern slides the technic of treating fractures of the vertebral column.

THE CHAIRMAN: Our next speaker is a man who, by his own achievements and his ability to give others the benefit of his knowledge, has become a true leader in the realm of traumatic surgery — the clinical professor of surgery and visiting surgeon of the New York Post Graduate Medical School and Hospital, Dr. John J. Moorhead. His paper is entitled "The Present Status of Traumatic Surgery."

THE PRESENT STATUS OF TRAUMATIC SURGERY

Abstract

JOHN J. MOORHEAD, M.D.*

NEW YORK CITY

TRAUMATIC surgery is one of the later developments in this branch of medical science, despite the fact that treatment of the injured was the earliest type of surgery.

Wounds. It is conceded that every wound not made with surgical intent is already infected, and to that degree every accidental wound is a problem in the treatment of infection. The main factors are the source, site and size of the wound, and the elapsed time since it was sustained. This means that a superficial small wound may be more dangerous than a deep large one. In all forms of traumatology, the first six hours post-trauma might well be called the "golden period." Injuries treated during that interval are generally free of complications and are assured of speedy repair. The treatment of wounds revolves around methods of sterilization, drainage and suture. There are only two ways to sterilize a wound: mechanical and chemical. The first is the safer and surer, and it has been abundantly proved that simple methods are more effective than complex ones. Soap and water in liberal doses make the best cleansing agency, since our aim is to rid the wound of all foreign material. The wound should next be loosely closed with interrupted nonabsorbable suture, with a small drain left in place. For the larger wounds, or for those with devitalized tissue, a more rigorous cleansing is followed by removal of all the bruised and crushed tissue. We have accomplished this satisfactorily if the parts bleed, healthy color is apparent, and the muscles contract. This debridement should not be sacrificial, and there is usually no necessity for removing large segments of the wound; it is a shaving-off process and not a gouging-out mutilation. When the bleeding has stopped, a final soap and water cleansing is given in the case of a large wound, and interrupted sutures are placed, but are left untied until the third day. I am convinced that our morbidity and mortality from wounds would be cut in half if the "golden period" of attack were observed and the method of secondary suture were more universally employed. Tetanus antitoxin, with or without gas-gangrene serum, should be given in all soil-contaminated wounds, and in those involving the buttocks or regions where the musculature is thick.

Infected wounds. Infected wounds are no longer subjected to incision and drainage unless there are localized fluctuation, localized tenderness and localized induration. Most infected wounds can be aborted, and many can be localized by continuous, hot, wet dressings. Incision for the release of pus should be long, and deep enough to be self-gaping. The approach should be vertical and in the direction of the main underlying structures.

Joints. Synovitis and bursitis are best treated by early aspiration and early use, especially where the knee is involved. Atrophy of the quadriceps muscle occurs very promptly in any knee injury, and the patient should accordingly be instructed to shrug the

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*Professor of clinical surgery, New York Post Graduate Medical School.

kneecap and practice lifting the extended leg. In all joint injuries early use is more preferable than prolonged splinting.

Burns. The main considerations in the treatment of burns are relief of pain, avoidance of shock and infection, and prevention of deformity.

Tendons and nerves. If immediate suture of lacerated tendons or nerves is impracticable, they should be tagged for identification. Secondary operation can be done in from ten to fourteen days in clean wounds, but not for several weeks in infected ones. Only fine silk sutures should be used for coaptation. Do not despair of nerve suture, for many months may be required for restoration of function.

Dislocations. After immediate reduction the problem is to restore function during the period of capsular repair.

Fractures. In the fracture lexicon there are four R's, namely recognition, or differentiation; reduction, or setting; retention, or splinting; and rehabilitation, or functioning. Anesthesia should be used oftener as an aid to reduction, and for this purpose evipal given intravenously is an effective agent. After two vain attempts to effect reduction by manual means, some sort of mechanical reduction usually becomes imperative. Open correction is now required less often and will soon be limited to fractures resistant to other methods. It is still, however, the treatment of election in fractures of the patella, the olecranon and the condyles. For compound fractures of the puncture type, soap and water cleansing and immediate splinting suffice. For others, the most rigorous soap and water cleansing is needed, followed by debridement and primosecondary suture. Splintage should first of all be safe, next simple, and lastly serviceable. Beware circular casings in fresh fractures, and do not neglect to pad bony prominences.

Physiotherapy. Physiotherapy has a place in traumatology, but its best form is self-use. The practice of advocating unlimited baking and massage is often unwise, as it teaches the patient to rely on outside aid and encourages a mental state that defeats personal activity. Physiotherapy in the form of self-use should begin early, and should be coincidental and not a terminal form of treatment.

Antiseptics. Any traumatic surgeon who seeks to substitute a flaming antiseptic for soap and water is a discredit to his profession. Practice traumatic and not chromatic surgery.

The chief aim of traumatic surgery in the acute stage is the relief of pain and of shock and the prevention of infection. These, in turn, in the later stages lead to the prevention of the three D's of traumatology — death, disability and deformity. Traumatic surgery, like general surgery, sometimes encounters insurmountable hazards, and we are reminded that we are confronted by three general groups of patients: (1) those who die independently of what is done for them; (2) those who get well independently of what is done for them; (3) those who get well on account of what is done for them. It is to the third group that I have directed your attention in an attempt to sketch some of the accepted precepts that should guide us in our efforts to limit the toll of the accident problem.

115 E. 64th Street.

DISCUSSION

THE CHAIRMAN: Dr. Moorhead's most interesting paper will be discussed by Dr. Allen G. Rice, visiting surgeon to the Springfield Hospital.

DR. ALLEN G. RICE, Springfield: Dr. Moorhead's address has been no dry, professional lecture that taxes your attention, but a sparkling talk that will be remembered. Dr. Moorhead is not only a master of his subject, but he uses the English language with such charming diction and delightful phrasing that one cannot help but remember his points.

I still like to use the word "contaminated" as well as "infected." It seems to be that in the first six hours the wound is contaminated; I wonder if it is really infected. To me, infection means that the germs are not only there, but to a certain extent have penetrated cells and tissues and have become true parasitic organisms. In this stage it is impossible to clean the wound mechanically with soap and water, whereas previously, when the wound is only contaminated, it can be done. I am glad to hear Dr. Moorhead bring back good old soap and water, for after all asepsis is cleanliness, and soap and water is the first cleansing agent and also the best.

Dr. Moorhead ended with a plea for certain gifts that would bring to him the traumatic surgeon's heaven. It would be the general surgeon's heaven, too. I should like to pray for one other gift. Most traumatic surgery is bound up with automobile accidents or industrial accidents; these cases all involve judicial red tape and legal technicalities of one kind or another that do not always work out to the advantage of the doctor. Why should it be made difficult for the man who saves lives, prevents deformity and shortens disability to be appreciated and properly compensated?

THE CHAIRMAN: As a nominating committee for next year's officers I appoint Dr. Halbert G. Stetson, of Greenfield, Dr. John W. O'Meara, of Worcester, and Dr. Mark H. Rogers, of Boston.

The next speaker is a man whose wide experience in the handling of the results of traumatism, combined with close association with a large insurance company, renders him especially well qualified to discuss the industrial insurance aspects of traumatic surgery. Dr. Joseph H. Shortell, visiting surgeon of the Boston City Hospital, will speak on "The Industrial Insurance Aspect of Traumatic Surgery."

THE INDUSTRIAL INSURANCE ASPECT OF TRAUMATIC SURGERY

JOSEPH H. SHORTELL, M.D.*

BOSTON

THE Industrial Insurance Act, more generally known as the Workmen's Compensation Act, went into effect on July 1, 1912. This law was designed to help men injured in industry. Previous to its enactment, an injured employee was obliged to sue his employer, and in that suit he had first to prove that the employer was negligent. He had then to prove that he himself was in the exercise of due care, that he was not injured by the negligence of a fellow employee, and also that he had not assumed the risk of the injury. The last three are called the common-law defenses, of which the employer could take advantage. When the employee did have an actionable injury there was a long delay with heavy legal expense, and the net results were quite unsatisfactory to all concerned.

For this reason the act was enacted by which employees injured in industry could be compensated. When it first took effect its enforcement was naturally conservatively handled, for the primary intention was to establish it. Two weeks was the limit for medical attention, and at the end of that period the entire medical expense fell on the injured man. At that time compensation did not begin until two weeks after the injury, when the injured employee received one half his weekly wage, not to exceed ten dollars a week.

When the act was first adopted the medical provision was as follows: "During the first two weeks after the injury the insurer shall furnish reasonable medical and hospital services and medicine when they are needed."

In 1914, under Chapter 708, Section 1, this provision was stricken out and the following section enacted to cover the medical provisions:

Section 5. During the first two weeks after the injury, and if the employee is not immediately incapacitated thereby from earning full wages, then from the time of such incapacity, and in unusual cases, in the discretion of the board, for a longer period, the association shall furnish reasonable medical and hospital services, and medicines, when they are needed. Where, in a case of emergency or for other justifiable cause, a physician other than the one provided by the association is called in to treat the injured employee, the reasonable cost of his services shall be paid by the association, subject to the approval of the Industrial Accident Board. Such approval shall be granted only if the board finds that there was such justifiable cause and that the charge for the services is reasonable.

Presented at the annual meeting of the Massachusetts Medical Society, Boston, June 3, 1937.

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In 1917, through Chapter 198, the statute was amended by providing that the employee might select a physician other than the one provided by the insurer, as follows:

Section 30. During the first two weeks after the injury, and, if the employee is not immediately incapacitated thereby from earning full wages, then from the time of such incapacity, and in unusual cases, in the discretion of the department, for a longer period, the insurer shall furnish adequate and reasonable medical and hospital services, and medicines if needed. The employee may select a physician other than the one provided by the insurer; and in case he shall be treated by a physician of his own selection, or where, in case of emergency or for other justifiable cause, a physician other than the one provided by the insurer is called in to treat the injured employee, the reasonable cost of his services shall be paid by the insurer, subject to the approval of the department. Such approval shall be granted only if the department finds that the employee was so treated by such physician or that there was such emergency or justifiable cause, and in all cases that the services were adequate and reasonable and the charges reasonable. In any case where the department is of the opinion that the fitting of the employee with an artificial eye or limb, or other mechanical appliance, will promote his restoration to industry, it may order that he be provided with such an artificial eye, limb, or appliance, at the expense of the insurer.

In 1927, under Chapter 309, this section was amended by including the words: "cases requiring specialized or surgical treatment," and by adding the words: "together with the expenses necessarily incidental to such services"; so that the section read as now embraced in Chapter 152 of the General Laws:

Section 30. During the first two weeks after the injury, and if the employee is not immediately incapacitated thereby from earning full wages, then from the time of such incapacity, and in unusual cases, or cases requiring specialized or surgical treatment, in the discretion of the department, for a longer period, the insurer shall furnish adequate and reasonable medical and hospital services and medicines if needed, together with the expenses necessarily incidental to such services. The employee may select a physician other than the one provided by the insurer, and in case he shall be treated by a physician of his own selection, or where, in case of emergency or for other justifiable cause, a physician other than the one provided by the insurer is called in to treat the injured employee, the reasonable cost of his services shall be paid by the insurer, subject to the approval of the department. Such approval shall be granted only if the department finds that the employee was so treated by such physician or that there was such emergency or justifiable cause, and in all cases that the services were adequate and reasonable and the charges reasonable. In any case where the department is of the opinion that the fitting of the employee with an artificial eye or limb, or other mechani-

cal appliance, will promote his restoration to industry, it may order that he be provided with such an artificial eye, limb or appliance, at the expense of the insurer.

This covers the law as it stands except for the amendment of Chapter 164 of the Acts of 1936, of which I shall speak later.

We must consider it from three angles: first, from the standpoint of the injured employee, because after all he is the most important; secondly, from the point of view of the doctor; and thirdly, from the point of view of the employer.

When the law was first enacted (this was some years before I was in practice), I am told that the companies listed certain doctors' names as the prescribed doctors. Too often, unfortunately, they were selected not for their ability but for what it would cost. Industry soon found that this method was wrong, and so effected the changes here outlined. In 1914 the words "in unusual cases for a longer period in the discretion of the department" were added.

Many cases were in dispute, among them that of Galen Moore (225 Mass. 535), which was held to be not unusual within the meaning of the statute, because the employee was able to go to the doctor's office for treatment. It was probably this case which caused the legislature in 1927 to add the words "or cases requiring specialized or surgical treatment." In this connection an interesting case is the Meuse Case (262 Mass. 95), in which the court said: "The legislature has not made the decision of the question whether the case is unusual dependent upon the probability of restoration of the employee's ability to work." This case came before the Supreme Court again in 1930 (270 Mass. 29), at which time the employee had received the maximum amount of compensation due under the act, and it was held that the words "for a longer period" as contained in Section 307, gave no right to the board to require the insurer to pay for medical and hospital services after the statutory compensation period had passed, and after the insurer had paid in full the compensation to which the employee was entitled. This would not be true at the present time in cases that are considered those of total permanent disability, because of the amendment that went into effect in September, 1935, under which the employee may get compensation for the remainder of his life provided he stays so disabled, and during this period he would be entitled to medical attention, if necessary.

Several other suits may be mentioned, among them the Bourgardes Case (265 Mass. 449). This concerned a claim under the "unusual cases" clause of the act. The employee was injured on January 14, 1926, and the court found that the new amendment of 1927 was not applicable. In the Souza Case (269 Mass. 441) the matter of contention was hysterical paralysis on the right side. This was decided to be not an "unusual case." The Bean Case (275 Mass. 543) was also decided as not an unusual medical one. This was a case with rupture of the urethra, and sounds had to be passed every ten days. The employee was able to work. It was ruled that the doctor was not a party to the employee's claim, and that he must petition for himself; the doctor attempted to appeal the decision. In the Panasuk Case (217 Mass. 589) the employee was unable to speak or read English; no instructions about medical attention were given to him. It was held that he had the right to choose his own doctor. In the Pappas Case (262 Mass. 51) the court ruled that the term "unusual" was not a proper subject for opinion evidence.

In the Allen Case (265 Mass. 490) the doctor was on the hospital staff, and it was held under the rules of the Industrial Accident Board that he was not entitled to charge for his services. We might continue indefinitely quoting cases which have occurred under the medical provisions. An important recent suit was the Bruso Case (1936 — A. S. 1911), decided on October 28, 1936, on a phase quite interesting to the insurance companies. It was held that in a third-party settlement medical benefits are part of the compensation, and the insurer is entitled to retain out of the proceeds of the settlement what he paid out.

The amendment of June, 1936, removed the words "and if the employee is not immediately incapacitated thereby from earning full wages, then from the time of such incapacity." It was thought when the decision in the Picanso Case (281 Mass. 476) was rendered that what moved the Supreme Court to its decision were the words I have just quoted. The court seemed to feel that the employee should be incapacitated before he was considered entitled to medical attention. I think that the elimination of these words will overcome the decision in the Picanso Case, which concerned dental work done after the first two weeks, where the patient lost no working time from incapacity, and where it was held that the services were not to be paid for by the insurer.

The provision for artificial eye or limb or other mechanical appliance was added in 1930. In a case where the department is of the opinion that the fitting of the employee with such an appliance will promote his restoration to industry, it may order that he be provided with it at the expense of the insurer.

Impartial doctors play a most important part in the administration of the act. Section 9 provides that "the department or any member thereof may appoint a duly qualified impartial physician to examine the injured employee and to report. . . .

The report of the physician shall be admissible as evidence in any proceeding before the department or a member thereof: Provided, that the employee and the insurer have seasonably been furnished with copies thereof." This is covered in the Emma Case (242 Mass. 408), where the court said that the impartial report may be offered in evidence provided that the employee and the insurer have seasonably been furnished with copies thereof. The report may be objected to if it is based on history obtained from a third party and not from the employee.

The injured employee's refusal to accept operation has been brought up many times. This is covered by the Supreme Court ruling in the case of Floccher (221 Mass. 54) which states that it must be shown, first, that the employee is a fit subject to be operated upon and, secondly, that the result of the operation will be a real and substantial physical gain. In the Snook Case (264 Mass. 92), the court held that the willingness to submit to the operation was not a test for its performance, but the right to guard life and limb from unreasonable peril; and that it was an employee's duty to submit to such an operation when the existing incapacity and suffering could be lessened without serious risk to life or member.

The medical section of the Massachusetts act is very liberal. The two weeks' medical attention allowed originally was found to be highly unsatisfactory, not only from the standpoint of the employee's having to pay the expenses, but because of the employee's medical neglect, since he was cast adrift at the end of two weeks and his injuries many times became very serious and caused prolonged periods of disability. The insurance companies saw this early and began to give medical attention irrespective of the limitation placed upon their liability by the statute. They found this practice more economical in the long run, as it lessened the period of disability; and they considered it more in the spirit of the times and of the act.

Section 9-B was added by the legislature in 1935; it constituted a radical departure from any previous provision. It calls for the appointment of three physicians to be known as industrial disease referees, in all claims involving an industrial disease. Their report is filed with the Industrial Accident Board, and their diagnosis is binding on the parties and the board.

The insurance companies have learned a lesson from the early stages of the act; namely, that poor medical services are expensive. In recent years they have furnished injured employees with the best possible medical and surgical attention that can be obtained; this statement is made advisedly. Particularly is this so in regard to surgery. Some wonderful operations have been performed and cures brought about by the good surgical technic of the doctors engaged. There is undoubtedly full medical attention during the entire period of the employee's need of it. This practice is followed not only because it is humanitarian but because it is good economy on the part of the insurer for the employee to get well as quickly as possible.*

This account summarizes the act as regards industrial surgery. But I know that doctors are more interested in its application. Many of them feel that the fees paid under the act are inadequate. The rules of the board were adopted years ago on the recommendation of a committee of doctors, which it called together to assist in administering the law. The board has never adopted any fee table. In case of dispute the medical adviser rules on the reasonableness of the charges; the final decision, however, rests with the board. Previous to the enactment of the act, a large percentage of cases were treated both privately and in the general hospitals without any doctor's receiving a fee. Before the depression many doctors stated that they took no compensation cases. I myself have done industrial accident work since I first went into practice, and have found it interesting as well as profitable. Many times, doctors feel bitterly toward insurance companies; this is understandable in view of the fact that whatever right of action an injured man has against a doctor passes to the insurance company; he cannot maintain an action in his own behalf. The case of Vatalaro *vs.* Thomas (262 Mass. 383) illustrates this point. I know of many cases where a lawyer representing an employee has attempted to bring suit against the attending physician for malpractice, knowing that his client had no right to bring suit except through the insurance company. While the insurance companies, on the other hand, realize that a successful suit in such instances would enable them to regain their entire outlay for both medical attention and compensation, I have learned of only one instance in the twenty-five years of the act where they have resorted to legal action.

As regards fees, it is thought that they are too low; but I think that they are reasonable, for the most part, considering the amount of work which has been done. Most of the trouble between the doctor and the insurance company has to do with the bill. There is much to be said on both sides. When I first worked for the insurance companies I was an ardent champion of the doctors, even though a flagrant case of overcharging was pointed out to me. In that instance there were two doc-

*For the above summary I am indebted to the Honorable Joseph A. Parks, chairman of the Industrial Accident Board, and Mr. J. Frank Scannell, attorney for the Lumbermens Mutual Insurance Company.

tors (father and son) in attendance; the patient had a simple fracture of the tip of the fibula. This case could have been treated by almost any doctor, yet there was a charge of twenty-five dollars for the application of a cast by the son, and a charge for daily home visits for fourteen days, including Sundays, by the father. I could not see any reason why the injured could not have come to the doctor's office on crutches, or why he needed to be seen each day.

One also sees many bills for treatment covering every day for a month except Sundays, when the doctor is out of town. We should naturally regard such a bill with suspicion. On the other hand, it is equally true that the insurance companies do not give proper consideration to bills. They seem to think that most of them must be cut down whether or not a fair charge has been made. A doctor who has seen his patient a legitimate number of times and makes a reasonable charge receives the same treatment as one who has paid daily visits in a simple case and charges accordingly. This holds true whether the doctor is a specialist or a general practitioner.

Industrial accident insurance has greatly benefited surgery. Having had to pay a considerable sum, the average insurance company makes a careful accounting and analyzes the results obtained. Just as in the United States Army during the World War when a supposedly simple operation for varicocele often resulted in a hydrocele and atrophy of the testicle, and the War Department passed an order against the operation, so the insurance companies have thrown their weight against fusion operations in acute fractures of the spine, and to a somewhat lesser extent in acute cases of sacroiliac sprain. Many doctors report cases when they first start to perform a given operation, but later state that they are opposed to it. Spinal fusions are still done in many acute cases, despite the present opinion against them. I remember in particular a compression fracture of the spine with an ileus obstruction. The obstruction quieted down in two or three days. A fusion operation was then done, with recurrence of the ileus, and the patient was operated upon for intestinal obstruction, the surgeon supposing that a loop of the bowel had been caught; yet no loop was found. It was a pure case of ileus caused from reflex action from trauma to the spine. Another instance is the fusion of the subastragalar joint in acute cases of fracture of the os calcis. This was formerly heralded as a cure-all, but today we find that most of those who study this condition are against such fusion as a primary procedure. This observation is not intended as a reflection against the work of doctors; we have only to point out the wonderful accomplishments of Doctors Davis and Rogers in their reductions of compression fractures of the spine, and those of Doctors Cotton and Hermann in fractures of the os calcis, in order to show the rapid progress made during the last few years.

The latest condition to cause serious concern is rupture of the intervertebral disk or nucleus pulposus. Doctors Mixter, Hodgson and Barr have done unusual work in this field. They are conscientious men and are very careful in their selection of cases, whereas many other surgeons are inclined to be radical and to utilize any new diagnosis or method to their own advantage. A copy of a diagnosis by x-ray on a patient outside of Massachusetts, dated September 24, 1936, reads as follows:

> Lumbar thoracic spine shows a rupture of the intervertebral disks of the tenth, eleventh and twelfth dorsal vertebrae and the first, second and third lumbar vertebrae, with a prolapse of the nucleus pulposus through the cartilaginous plate into the adjacent spongiosa of the contiguous vertebrae. The prolapse is surrounded by definite sclerotic layers of bone. The loss of this nuclear substance, which serves as a shock-absorber and a hydrodynamic ball-bearing for the spine, results in the two vertebral bodies' being brought together, with the production of bony changes, resulting in chronic back pain. In these cases if the prolapse extends posteriorly, as it seems to have done in this case, neurologic symptoms result from pressure of the prolapse on the cord. This leads to permanent cord change. The duration of this condition as shown in these films is that of at least four or five months, and possibly more.
>
> The prognosis is not good. There is some degree of functional back motion that is lost forever, and more or less continual back pain usually results. Treatment is directed toward the limitation of back motion and is therefore of necessity long and often unsatisfactory. This case would necessitate the application of a plaster cast or the use of a spine brace, to be worn for many months. . . .

These x-rays were later examined by several Boston doctors, and showed only an old growth condition of the epiphysis. There was nothing to indicate rupture of the nucleus pulposus with pressure on the spine. This case is cited not as evidence that such rupture never occurs, — for I have seen it, — but as an illustration of the fact that when a new operation and a new diagnosis are developed, many surgeons at once conclude that each case with somewhat similar symptoms is of this sort, and proceed to operate. This not only causes a loss to the insurer, but may cause permanent damage to the employee.

The Workmen's Compensation Act in Massachusetts appears adequate. It has been carried out by competent men, with the interest of the injured man always at heart. The matter of fees should be straightened out by the board, the Massachusetts Medical Society and the insurance companies. As regards fads in operations, I should like to give a warning against radicalism, and to express the hope that the members of the society will follow conservative treatment for these injured patients.

464 Commonwealth Avenue.

Discussion

Dr. Shortell: There are one or two questions that might come up in discussion, and I think it would be quicker to answer them now. The following questions have been asked of me.

1. Is there anything the attending doctor should do to be sure that the patient's compensation is properly safeguarded?

Yes. First of all, he should be sure that it is reported if he thinks it is due to injury or disease sustained while at work. Secondly, if the case is an open-and-shut one, and if the insurer does not see fit to pay the injured man his compensation, he should be advised to go before the Industrial Accident Board and ask for a hearing. It is not necessary for him to employ legal aid, because the Commissioner will act as his adviser; and many times the man is just as well off without an attorney. If, however, the case is complicated or involves death, so that legal problems are certain to arise, the man or his relatives should consult an attorney who is familiar with the act; if this is beyond his means, the free services of the Legal Aid Society can always be obtained.

On the other hand, many doctors start cases where there is no basis for a claim. A man rides on a truck and gets hemorrhoids. He says to a doctor, "Don't you think the hemorrhoids resulted from riding on the truck?" The doctor says, "Yes, I think so." Then a hearing is started before the board; but the doctor will not tell the board what he told the patient. In many cases a man who has been wheeling out ash barrels gets appendicitis and says to a doctor, "Don't you think that is what gave me appendicitis?" The doctor says "Yes," and the man brings a claim; but the doctor will never so testify to the board. With infected glands in the groin the same sort of thing happens.

2. What is the present compensation?

It is two thirds of the average weekly wage, not to exceed eighteen dollars a week. Until about a month ago the worker got no compensation for the first full week until he had been out a month. Now, I believe, he has to be out only two weeks in order to collect for his first week.

3. If a case promises to be prolonged, is it necessary for the doctor to notify the insurance company to that effect?

I do not know that it is; but it is important that the insurance companies know whether the case is mild or serious. For exactly as in automobile accident insurance, an adequate reserve must be set aside. If a case is reported as a minor one, with a probable absence of three or four weeks, no great reserve is set aside; whereas if a man has an injury that is certain to keep him out a year or two, it is necessary for the insurance company to place in reserve enough money to cover all possible expenses, for both medical and compensation services.

4. Do "unusual" cases have any significance at present?

I think I have answered that by saying that the "unusual" part does not matter. Many doctors still think they are limited to two weeks' treatment; this is not so, for the law reads "not only in unusual cases, but in cases requiring specialized or surgical treatment."

The Chairman: In order to deal adequately with the most essential factors in the tremendous problem of fractures, we have reached out to Chicago to secure our next speaker. I know of no one who has had a broader experience and possesses a greater ability to present clearly the subject of fractures than the visiting surgeon at the Presbyterian Hospital at Chicago, the clinical professor of surgery at Rush Medical College, Dr. Kellogg Speed. His paper is entitled "Untoward Results in the Treatment of Fractures."

UNTOWARD RESULTS IN THE TREATMENT OF FRACTURES*

Abstract

Kellogg Speed, M.D.

CHICAGO

THE speaker prefaced his remarks as follows: "We all, as practitioners, get into difficulties at times with fractures. If you consider the causes some cold, dark night and sit down and try to analyze just what has gone wrong in a series of cases, you may come to such conclusions as I have made."

Among untoward results he mentioned angular deformities, loss of length in a bone, infection causing osteomyelitis in uncomplicated fractures, involvement of blood vessels, nerves, tendons or muscles with unfortunate end results, disability in the joints in fractures in and about the joints, delayed-union or nonunion cases, and neurasthenic states caused by coddling a patient so much that he is disinclined to return to work. Elaborating on the various common types of fractures of both upper and lower extremities, fully illustrated by lantern slides, the speaker brought out most emphatically the do's and don't's of up-to-date fracture therapy.

*By special request of Dr. Speed, the complete paper is not presented.

Discussion

The Chairman: The discussion of this most enlightening paper will be given by Dr. Charles E. Ayers, visiting orthopedic surgeon at the Memorial Hospital, Worcester.

Dr. Charles E. Ayers, Worcester: I agree with everything that Dr. Speed has said. The things that impressed me most were the complete study of the patient and x-rays. I think we have all been guilty in not carrying through our part in some of our cases. I have had some difficulty with supracondylar fractures, where on examination the pulse at the wrist was not present. I am always afraid of this type of case, and I prefer to put a Kirschner wire through the ulna near the olecranon and suspend it in the overhead position until the pulse returns.

The Chairman: We should like to have the report of the nominating committee.

DR. HALBERT G. STETSON, Greenfield: The committee nominates Dr. Leland S. McKittrick, of Boston, as chairman for the coming year, and Dr. John M. Fallon, of Worcester, as secretary.

The nominations were accepted, and the secretary was instructed to cast a single ballot for the election of Dr. McKittrick and Dr. Fallon.

THE CHAIRMAN: The Sixth Surgical Service of the Boston City Hospital receives the great mass of the traumatisms of this, New England's largest city. The chief of this service, Dr. Otto Hermann, who has had an unusual opportunity to study compound fractures, will present the next paper, entitled "The Treatment of Compound Fractures."

THE TREATMENT OF COMPOUND FRACTURES

Abstract

OTTO J. HERMANN, M.D.*

BOSTON

EVERY compound fracture presents specific problems of what to do and when to do it. Yet a fairly routine treatment up to the reduction and fixation point is an aid to expeditious work. From that stage on, the treatment varies according to the surgeon's judgment and experience. The treatment of all fractures should begin at the scene of the accident. With efficient first-aid treatment, shock is minimized by hemostasis, traction, fixation, early administration of hypnotics and proper transportation. Unfortunately in our city the first-aid treatment in such cases and the subsequent transportation are quite crude. Most of the cases are delivered at the Boston City Hospital by the police ambulances so that our police should be trained in the modern first-aid care of fractures.

At present, then, treatment begins on the accident floor of the hospital. There a rapid but careful examination is made, with special search for such major complications as shock, brain injury or trauma to any important viscus. The surgeon treating compound fractures must not only be expert in that field, but must be capable of handling any other important results of traumatism.

Simultaneously with this examination the initial treatment is carried on. If the patient is in a moderate degree of shock, heaters and blankets are used and morphine is administered; a rapid exposure of the wound is then made. After the removal of gross debris, the wound is covered with a sterile dressing. Clothing is removed, and a temporary traction splint is applied. An x-ray examination is then made, and if the patient's condition is satisfactory he is taken to the operating floor. If, however, the patient is in severe shock he is at once transported to the shock room where adequate treatment is instituted. In addition to the conventional method of shock treatment we have for the past few years treated shock by hourly intravenous injections of 50 cc. of sodium bicarbonate solution, prepared according to the formula and method advocated by Dr. Kenneth Coonse of the Boston City Hospital. If there is shock with hemorrhage, a transfusion is given. In all cases, after a careful testing, a prophylactic dose of gas-gangrene antitoxin and tetanus antitoxin is given.

When the patient has recovered from shock, he is taken to the x-ray room and then to the operating floor. After he has been anesthetized, a most thorough cleansing of the wound and the surrounding operative field is done. The wound is then carefully debrided, with special attention to the removal of all devitalized tissue and to the avoidance of unnecessary trauma to the healthy tissues. This is all done with instruments, without touching the wound even with the gloved finger. Instruments are frequently changed to avoid carrying contamination to the freshly debrided wound. Complete hemostasis is carried out to avoid the presence of blood clots, which encourage infection. Following the debridement the wound is gently and moderately irrigated with salt solution, followed with a mild irrigation of alcohol, then with a mercurochrome-acetone-alcohol solution, hydrogen peroxide or ether. During the past five years we have found that the incidence of sepsis was much greater in cases irrigated by salt solution alone and in goodly quantity than in those treated by a moderate salt solution irrigation plus chemical antiseptic solutions. When such chemicals are used, extreme care is exercised in avoiding as much as possible the periosteum and the bone.

The surgeon changes gown and gloves and redrapes the operating field. With an entirely fresh set of instruments he treats the fracture proper. The bone ends are gently curetted to remove all possible contamination. The instrument is discarded and the wound gently and moderately ir-

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*Surgeon-in-chief, Sixth Surgical (Bone and Joint) Service, Boston City Hospital.

rigated. All bone chips that are not hopelessly devitalized or contaminated are preserved.

From this point on, the treatment will accord with the surgeon's experience and judgment. We realize that some men will proceed exclusively along conservative lines; that is, they will never plate, band, suture or nail this type of fracture. We unhesitatingly do any of these things if we think the case requires it. Because of improved methods of closed fracture treatment, however, it is found that only a small percentage of these cases require internal fixation. Owing to the relatively high incidence of delayed union and nonunion, accurate reduction and apposition are paramount. To maintain these, internal fixation is in some cases sorely needed. We have found these cases remaining free from sepsis and giving good end results relatively early; the presence of metallic foreign bodies has not necessarily increased the incidence of sepsis.

We come now to another controversial stage of compound fracture therapy. Shall the wound be left open and treated by the Orr method, or shall Dakinization, primary suture and drainage, or simple primary suture be used? Each method has its many advocates, and each is good. The surgeon should use the procedure which has given him the best results. In many cases, however, there will be no doubt of the superiority of one method over the others. Our preference is for primary closure without drainage; we have used it in roughly 75 per cent of our cases. In these instances the wound is sutured very loosely so that there will be ample opportunity for tissue drainage between the sutures. If the soft tissues cannot be readily closed over the bone without tension, we do not hesitate to make lateral incisions to relieve this tension.

The final step in this initial compound fracture treatment is the application of external fixation or traction. Those cases in which internal fixation has been applied usually need only a simple plaster of Paris cast or moulded splint. Most of the others will require traction, probably in a Thomas's splint suspended from a Balkan frame.

One type of compound fracture differs from the common type which we have been discussing. This is the pinhole or puncture type. It has been said that 75 per cent of such wounds heal immediately. However, we have decided that all such fractures should be treated by the regular excision and debridement method.

There are a few cases where a compound fracture is so mangled that amputation is the only surgical solution. In the borderline case we must not forget that attempting to save the limb may involve virulent infection endangering life, and also that even if the limb is saved, useful function is unlikely.

Despite the meticulous care given to compound fractures, sepsis and other complications may occur. Gas-bacillus or tetanus infections should receive specific serum treatment as well as adequate local treatment. Streptococcus and staphylococcus infections should receive blood transfusions, preferably well-placed fractional ones, as well as free local drainage. Delayed union and nonunion are most frequent in compound fractures. They can be minimized by careful attention to hygiene, by physiotherapy and by the indirect stimulation of weight-bearing through the use of ambulatory splints. Many cases of delayed union are saved valuable months of convalescence by some operative procedure such as multiple drill holes, an inlay graft, a chip graft or an osteoperiosteal graft.

SUMMARY

1. There should be intelligent first aid in compound fractures: proper handling, modern emergency splinting and careful transportation.

2. It is absolutely imperative to consider the patient first and the fracture second. Be prompt in giving proper rest, stimulation, prophylactic gas-gangrene and tetanus antitoxin injections and general supportive treatment, with an emergency dressing and external traction and splinting. Also be careful in the general examination to look for signs and symptoms of internal damage.

3. To ensure good teamwork, have well-planned methods of skin preparation, debridement and wound cleaning.

4. In the treatment of the fracture itself, be open-minded, size up the situation quickly, and be ready to use any of the several accepted methods of traction, internal fixation, and so forth.

5. Do not be dogmatic about "closed" and "open" treatment. Be ready to use either according to your judgment.

6. Choose carefully and well the type of external fixation.

7. Counsel wisely with others in deciding between primary amputation and probable later amputation for complicating virulent sepsis, with its likely fatal result.

8. In the treatment of later complicating gas-gangrene or tetanus infection, give prompt and radical treatment with appropriate stimulation and supportive medical treatment. The same can be said of the virulent streptococcic and staphylococcic infections.

9. Consider bone-grafting as a valuable economic timesaver in carefully chosen delayed-union cases.

520 Commonwealth Avenue.

Because of lack of space this paper has been partly abstracted. It will be published in toto in the reprints.

Discussion

The Chairman: Dr. Hermann's paper will be discussed by Dr. Ernest M. Daland, visiting surgeon at the Massachusetts General Hospital.

Dr. Ernest M. Daland, Boston: On several occasions I have reviewed the cases of compound fractures at the Massachusetts General Hospital, and I have data on over 400 cases treated during the past fifteen years. This hospital does not maintain an ambulance service for accident cases; hence we do not see nearly so many compound fractures as Dr. Hermann does at the Boston City Hospital. He doubtless sees a larger percentage of serious compound fractures from industrial and automobile accidents than we do in our clinic.

In many of the larger cities of the United States the police and firemen are being instructed in first aid and transportation for patients with fractures. I am told that no city or town in Massachusetts offers this training. The state police do receive it, and the Red Cross first-aid stations also have personnels trained in handling such cases. It is essential that people who care for these patients should be taught to apply proper first-aid dressings and to "splint 'em where they lie."

During the last few years certain men have advocated the use of steel plates or bands in the treatment of compound fractures. Until about three years ago this practice was frowned on at the Massachusetts General Hospital. Since 1934 I find that plates or bands have been used in 13 cases. There was sepsis in 5 of these cases, 2 patients developed osteomyelitis, and 1 case required amputation. I am convinced that there are instances where plates can be safely used, but they must be applied at the initial operation, and the operator, not his well-meaning friends who see the case a few days later, must decide whether to employ them. Our bad results came in cases where the wound was reopened a few days later and a plate was applied. The wound must not be closed, and may be treated by the Carrel-Dakin regime or by the application of a boric-ointment pack.

We must distinguish between compound wounds occurring from without and those occurring from penetration of bone fragments from within. Of the latter group, the simplest is the puncture wound of the skin. Often the bone fragments have caused the skin to rupture but the bone has not necessarily protruded through the wound. Dr. Hermann advocates debridement in every case. But I find that the usual practice in these cases at the Massachusetts General Hospital is to treat the wound with an antiseptic, excise ragged skin edges but not explore the fracture site, and leave the wound open. I am not going to advocate either method, but let us look at our results.

The first slide shows the distribution of 66 cases of compound wounds of the forearm. Thirty-seven were caused by indirect violence, and of these, 33 were puncture wounds. Twenty were due to massive external trauma, and 9 to lacerations or bullets.

On the second slide you will note that of the 33 puncture wounds only 1 became septic, and that sepsis was minor. I repeat that the general practice on these cases was to do a minimum amount of surgery.

The third slide shows the distribution of the 89 compound wounds of the leg into four classes. There were 30 puncture wounds and 36 cases of massive external trauma.

The last slide shows the method of treating the 30 puncture wounds. There was 1 case of sepsis in 17 where simple cleansing methods were used. There was no sepsis in 13 cases in which debridement was carried out.

The Chairman: The last speaker has not only made a most thorough study of burns, but has also made an important contribution to their early treatment. Dr. Robert H. Aldrich, of the Boston City Hospital, will read a paper entitled "Treatment of Burns with a Compound of Aniline Dyes."

TREATMENT OF BURNS WITH A COMPOUND OF ANILINE DYES

Robert H. Aldrich, M.D.*

BOSTON

In 1933 I[1] published a paper entitled "The Role of Infection in Burns: the Theory and Treatment with Special Reference to Gentian Violet." I tried to show that the toxemia of a burned patient was due not to the absorption of a burned protein or to a shifting of the water balance, but to an invasion of the body through the burned area by virulent forms of the streptococcus. At that time gentian violet seemed to be the best available means of counteracting infection. The burn was protected from all gram-positive organisms and sealed under an eschar; analgesia was obtained and fluid loss prevented. Gentian violet, however, has one weakness; it is not a specific antiseptic against the gram-negative organisms, and sooner or later these bacteria offer complications. For two years I sought some other antiseptic that had all the advantages of gentian violet plus a more powerful action against the gram-negative bacteria. I investigated the aniline and the azo dyes as well as the chloramides, and now present a report on the use of a combination of acriviolet and brilliant green — acriviolet itself being a loose chemical combination of acriflavine and crystal violet.

Before giving a detailed description of this form of treatment, I should like to stress again my theory of the role of infection in burns. When Firor and I began working with burns at the Johns Hopkins Hospital, there were two main theories concerning the cause of toxemia and death in burns. The first was that absorption of split protein in the

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burned area was responsible. Numerous investigators had reported many substances, isolated from the burned area, the blood and the urine of burned animals or patients. Some of these were histamine, pyridine, guanidine, ptomaines and primary and secondary proteoses. There had been no uniformity in the results obtained by different investigators. Among the advocates of this theory were Robertson and Boyd,[2] who claimed to have isolated primary and secondary proteoses by alcoholic extraction from burned skin. When this extract was injected into unburned animals, they exhibited signs of toxemia and shock. For a time this seemed to have settled the question. But Underhill and his co-workers[3] at Yale repeated the experiments of Robertson and Boyd, using the same technic, and discovered that it was the ethyl alcohol of their extract which caused the animal's toxemia and shock.

Underhill[3] then proved definitely that there was no absorption from either the burned area or the edema fluid around the burn by injecting five times the lethal dose of strychnine into the burns of experimental animals without obtaining any evidence of strychnine poisoning. He followed this up with injections of trypan and methylene blue into the burned area, and found on tests and from sections that these dyes were not absorbed from the burned area back into the body. If none of these three substances are absorbed it seems very improbable that some split protein should be absorbed in sufficient quantities to cause any toxemia.

Underhill[3] then showed that there is an enormous shifting in the water balance in burned patients, with resulting concentration of the blood. In animals a third-degree burn of one sixth of the body area brings about a loss of one half of the blood volume in thirty hours. This seemed to him to be the cause of the toxemia of burns, and he felt that the logical treatment would be one which kept the blood chemistry normal. Such a treatment consisted chiefly of intravenous injections of normal saline. A review of Underhill's cases indicates that while this form of treatment was very good in combating primary shock, on the third or fourth day signs of toxemia set in and his patients ran much the same course as those of other investigators.

If, then, there is no absorption from a burn back into the body, and if, even when changes in the water balance are prevented, the symptoms persist, What is the cause of the toxemia? Firor and I began our work with this question. Briefly, our procedure was as follows. A review of the literature revealed that no work had been done on the bacteriology of burns. Repeated bacteriologic studies of fresh burns were done, and revealed that for the first twelve hours the areas were virtually sterile, but that beginning around the eighteenth hour positive cultures could be obtained on all burns. The early culture reports showed a mixed infection, but after seventy-two hours the pus which covered all burns was almost a pure culture of beta hemolytic and gamma streptococci. In a recent letter from Cruikshank, of Glasgow, I have been informed that he has confirmed our work on the last two hundred burned patients entering the Royal Infirmary.

There was a definite correlation between the clinical picture and the amount of infection. As the streptococci multiplied, the toxemia increased in severity. The patients' charts and the bacteriologic reports went hand in hand. With this evidence, and with the awareness that burned patients had large, open surgical lesions bathed in virulent streptococcic pus, we saw no reason to search for some obscure split protein. Additional pieces of evidence that lent weight to our theory were these: in those patients who became very toxic, blood cultures were positive with the same strain of streptococcus that was found on the burned surface; in patients dying following a severe burn, the same germs as were found on the burn could be cultured from the heart's blood and the lungs. From this we concluded that the fundamental principle in the treatment of burns should be asepsis and antisepsis.

We tried various antiseptics in an effort to find a nontoxic agent with a high, specific germicidal power. Gentian violet was the outcome of this work. After its adoption we immediately began to get the results which we had expected. If a burn can be kept free from infection there will be no evidence of toxemia. In the two years following the introduction of gentian violet at Johns Hopkins Hospital, the mortality dropped from 42 to 13 per cent. However, gentian violet was not the ideal antiseptic, and contamination by gram-negative bacteria was a constant annoyance. No details of the gentian-violet treatment need be given now, inasmuch as it was used in exactly the same way as the new combination of dyes which I shall next discuss.

This combination of acriviolet and brilliant green is the most powerful antiseptic against the gram-positive bacteria that will not injure living cells; it also has a high specific action against the gram-negative organisms. In broth cultures it will not let gram-positive bacteria grow in dilutions of 1:1,000,000, or gram-negative in 1:10,000. In concentrations of 1:1000 it has a high phenol coefficient against all the pyogenic organisms. When applied to a burned area it very rapidly forms

a tough, flexible eschar which seals off the burn, sterilizing it at the same time. The eschar thus formed prevents fluid loss, and brings about analgesia by protecting the nerve endings.

The treatment of a severe, superficial, recent burn is divided into two parts. The first part deals with shock, and the second part with the burn itself after the patient is out of shock. The shock seen in a burn is almost identical with any surgical shock. The main difference is that it can be divided into three types. The type most frequently seen is the shock that comes on with or immediately after the burning. The patient presents the usual characteristics of rapid, thready pulse and low temperature and blood pressure; there is no change in the blood picture. By treating with heat, rest and fluids the shock can be overcome quite easily.

The next commonest type is the so-called secondary shock, or the histamine shock of Cannon, who believes it is due to the absorption of a histamine-like substance from injured tissue. This usually comes on from ten to twenty hours after the initial shock. The patient presents the same physical signs and symptoms just mentioned, but there is a change in the blood picture. There is an increase in viscosity of the blood. Taking 1.0 as the viscosity of water, normal blood has a viscosity of about 2.4, and in this secondary shock the viscosity increases to between 4.0 and 5.0; this is true of blood taken from capillaries and larger vessels. From my observations I feel that secondary shock is inadequately treated primary shock, and have never seen it develop when complete and prolonged initial treatment was instigated.

The third type of shock is fortunately not common, for it is usually a sign of impending death. It occurs about ten hours after the burn, and in patients who have not exhibited the initial shock. For some reason, probably such a sudden burning of the nerve endings that pain is almost nil, the patient skips the primary stage, and does not develop shock until rather late in the phase of water imbalance. The onset is usually rapid, presenting the typical picture, but a blood study here will reveal a viscosity of about 4.0 in the larger vessels and from 6.0 to 7.0 in the capillaries. This means that the patient's own red blood cells are acting as minute thrombi and emboli in all the capillary beds. This probably accounts for the degenerative changes noted in all the lymphoid tissue, and for the anuria and intestinal ulceration. This type of shock is hard to combat if allowed to develop, because fluids injected into the veins cannot be pumped out to thin the capillary blood.

All patients with a burn of one eighth or more of the body surface should be treated for shock immediately, whether or not it is present. The treatment of the burn should be delayed until the surgeon is certain that shock has been adequately combated or is not going to develop. This is the most important factor in caring for a burn, regardless of the technic used.

In the treatment of the burn itself, the simpler it is kept the better the end results will be. No preliminary cleanup is done unless the patient has previously been treated with an oil or ointment. This has to be removed before the aqueous solution of the aniline dyes can be applied. The blebs are excised, but no extensive debridement is done. The patient is placed nude under a cradle, and is sprayed with the dyes every hour by means of an atomizer. The eschar develops in about eight hours, and further spraying is unnecessary for the time being. The essential factor is the daily observation of the eschar. Unlike the tannic acid eschar, this one will not hide infection but softens and becomes moist if there is any underlying pus. If infection develops, the softened areas are excised, the underlying area is patted with a dry sterile sponge, and the dyes are reapplied. This technic is continued until epithelialization is complete or until granulating tissue has been formed to the point where it will accept skin grafts. If gross contamination of the eschar occurs, as frequently happens in burns around body orifices, the infected eschar can be removed with compresses of normal saline, and the dyes applied again.

When patients are treated in this manner they do not require nearly the amount of skin grafting that was formerly done. Even in what are apparently third-degree burns there are many islands of epithelium beneath the burned area at the base of the hair follicles which will grow and spread if they are not bathed in pus. The resulting scar is fairly soft, and does not give rise to many contractures.

In conclusion, there are two things to be emphasized. First, the treatment of burns is always difficult and involves a great deal of hard work; there is no assurance that mere spraying with these dyes will prevent mortality. Their use makes treatment easier, but in the combating of the shock, in the constant attention paid to the eschar and in the nursing care and the diet, great efforts are necessary. In patients with terrifically severe burns, blood transfusions should be used frequently to help combat infection and to give the patient added powers of healing. Secondly, while the new combination of dyes is as superior to gentian violet as gentian violet is, in my opinion, to tannic acid, it is not the final answer to all the problems presented by a burned patient. I am certain, however, that the conception of a burn as an infected surgical lesion is correct, and that it is infection rather than absorption of a split protein which causes death.

For, let me add again, where there is no infection there is no toxemia. If and when some substance better than the new combination of dyes is found, that substance too will be an antiseptic possessing the qualities of an analgesic and an escharotic.

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Discussion

The Chairman: The discussion will be opened by Dr. Richard H. Wallace, of the Massachusetts General Hospital.

Dr. Richard H. Wallace, Boston: Dr. Aldrich has covered the subject clearly and concisely, and there is very little to add. The treatment has been worked out very carefully by him. He has done a great amount of research work with his antiseptics, and has been able to improve them considerably over the period of the last three or four years. His statement that in the absence of infection there is no toxemia is a great truth. The smoothness of the convalescence of a patient with severe burns is inversely proportional to the amount of infection. The ideal treatment of burns, if we ever have it, will be one through which we are able to eliminate infection, and Dr. Aldrich has done much to minimize infection.

The Chairman: Dr. Merritt B. Low, of Deerfield, has been very much interested in burns, and has recently published an article with a little variation of treatment. He is a former resident surgeon at the Children's Hospital. Dr. Low, will you discuss Dr. Aldrich's paper?

Dr. Merritt B. Low, Deerfield: About two and a half years ago Bettman, on the West Coast, developed a new treatment of acute burns consisting in the application of 5 per cent tannic acid solution, followed immediately by 10 per cent silver nitrate. We started using this method at the Children's Hospital two years ago, and since that time have been greatly impressed by its effectiveness. Without going into detail in the routine treatment of shock, which is so important and which has been emphasized by Dr. Aldrich, I may say that we usually employ a general anesthetic, because the pain certainly adds to the shock. Then we carefully cleanse the burned areas with soap and water, peroxide, alcohol, or hexylresorcinol solution, and then apply the tannic acid, followed immediately by the silver nitrate, each being sopped on. An eschar forms within several minutes and dries forthwith, even to the point where a dressing or clothes can be put on without sticking. No further local treatment after this single application is necessary, at least for a number of days.

The important thing is to care for the general condition of the patient. We are much in favor of a continuous intravenous drip in badly burned children and babies, and the judicious use of blood transfusions is certainly indicated. I should like also to say a word in favor of plasma transfusions (with the red cells centrifuged away from sterile citrated blood). The serum protein is an important thing to follow, and this can be determined easily, owing to the development of the refractometer in the last few years. With this instrument, one drop of blood will give an accurate determination.

The main advantages of this method of treatment are these: it minimizes the local care necessary during the twelve to thirty-six hours required for successful application of an eschar using one of the dyes or tannic acid alone; and we get an immediate eschar, which, I must emphasize, remains dry. Thus we do much to combat shock and complications during those first twenty-four hours, which are so critical. Silver nitrate, of course, has a strong antiseptic value, and we avoid the contamination which is almost inevitable, at least in children, during the time the other sprays are being applied. The increased comfort of the patients is striking, and often within from six to ten hours they are feeling quite well, and in certain cases can be up and around. We also get a thinner eschar than with tannic acid alone. The thickness of the tannic acid eschar has often been bothersome.

This method is sometimes criticized on the ground that some viable epithelium may be killed through overvigorous treatment of the burned area, and that some extra keloid formation may be induced. I am not convinced that either of these objections is valid. The only bad keloids we have had were in a small baby where there was also keloid formation far in excess of normal in an unburned area which was the donor site of split-Thiersch grafts. The possibility of argyria, I believe, can be discounted; our silver is not "organic" or colloidal, there is only one application, and Underhill and others have shown there is very little absorption from the burned area anyway.

I have enjoyed Dr. Aldrich's paper very much. No doubt all such rational methods as his have their place in the treatment of burns. My feeling is that for very bad burns, where death seems imminent, or is inevitable unless we do something quickly and thoroughly, the tannic acid-silver nitrate treatment represents a valuable weapon, a distinct advance in therapy, and holds by far the best chance for the patient.

The Chairman: This meeting stands adjourned.

CHRONIC HEMOLYTIC ANEMIA WITH PAROXYSMAL NOCTURNAL HEMOGLOBINURIA

Study of the Mechanism of Hemolysis in Relation to Acid-Base Equilibrium

THOMAS H. HAM, M.D.†

BOSTON

THE clinical syndrome of chronic hemolytic anemia associated with paroxysmal nocturnal hemoglobinuria, first recognized as a disease entity by the two Italian investigators, Marchiafava[1] and Micheli,[2] has been reviewed recently by Witts[3] and Hamburger and Bernstein.[4] The present communication deals with the preliminary results of a study of the mechanism of hemolysis in three patients exhibiting this syndrome, of whom one* has already been reported as such by Hamburger and Bernstein.[4]

The blood picture of these three adult subjects, two males and a female, was characteristic of increased blood destruction, as evidenced by severe anemia, increased reticulocytes, elevated serum bilirubin and especially the constant finding of free hemoglobin in the plasma by spectroscopy. The plasma varied in color from light brown to reddish-brown. In two of the patients there was mild leukopenia and thrombocytopenia. Moderate splenomegaly was present in one. In the third patient,[4] whose spleen had been removed twenty-six months before these observations, the white blood-cell and platelet counts were normal or slightly increased. The bloods of all the patients belonged in Group O (International), and their red blood cells showed a normal fragility in hypotonic salt solutions. The Wassermann reactions were negative. At some time during the period of observation of each patient the urine appeared red, dark reddish-brown or almost black and contained large amounts of free hemoglobin, as demonstrated by spectroscopic examination. Variable amounts of albumin but no red blood cells were found in the urine. Hemosiderin, identified by the method of Cook,[5] was a constant urinary finding.

Quantitative determinations of the amounts of hemoglobin in the plasma and urine were made by a modification of the benzidine method of Bing and Baker[6] upon specimens obtained every three hours during day and night for periods of several days. In the two patients whose spleens had not been removed, an increase in hemoglobinemia was always observed during sleep, whether at night or during the day, and an increase in hemoglobinuria was frequently present. These phenomena did not occur if the patient was kept awake for twenty-four hours or kept awake under basal conditions in positions assumed during sleep. There was no apparent relation to the time or amount of ingested food or fluid. In the patient whose spleen had been removed, there was no increase in hemoglobinemia or hemoglobinuria during sleep, although definite nocturnal hemoglobinuria had been observed by Hamburger and Bernstein[4] before splenectomy.

Because of the elevation in the carbon-dioxide content of the arterial blood and the decrease in pH known to occur during sleep,[7, 8] it was suspected that a change in the acid-base equilibrium was related to the increased hemoglobinemia of two of the patients during sleep. When two subjects were given 40 gm. of sodium bicarbonate daily the hemoglobinemia and hemoglobinuria decreased; withdrawal of the alkali produced an increase in plasma and urine hemoglobin. When a large single dose of ammonium chloride (10 or 12 gm.) was given, the hemoglobinemia and hemoglobinuria increased, and in one patient the pH of the arterial blood fell to 7.25. In one patient the pH of the arterial blood during natural sleep was 7.3, with a carbon-dioxide partial pressure of 42 mm. of mercury; the usual increase in hemoglobinemia and hemoglobinuria occurred. In the same twenty-four-hour period, however, hyperventilation in a Drinker artificial respirator during natural sleep caused a rise in the arterial-blood pH to 7.47, a fall in the carbon-dioxide partial pressure to 28 mm. and a decrease in plasma and urine hemoglobin.

It was noted that on standing for four hours at room temperature or in the incubator, samples of whole clotted blood, defibrinated blood and blood containing heparin as anticoagulant, all developed progressive hemolysis. At ice-box temperatures hemolysis was minimal. Comparable samples of the blood of normal subjects showed no significant hemolysis under these conditions. Equilibration of the patients' defibrinated blood or blood containing heparin with mixtures of oxygen and

This investigation was aided in part by a grant from the J. K. Lilly gift to the Harvard Medical School.

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*Study of this patient was made possible through the courtesy of Dr. William B. Porter, of Richmond, Virginia.

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carbon dioxide, such that the partial pressures of the carbon dioxide ranged from 4 to 40 mm. of mercury, produced immediate hemolysis roughly proportional to the carbon-dioxide tension. There was no apparent relation to the degree of oxygen saturation, since equilibration with oxygen or air alone caused no hemolysis and since the substitution of nitrogen for oxygen in the carbon-dioxide mixtures caused no significant increase in hemolysis. The addition to the serum of lactic acid in a 10 or 20 millimolar concentration likewise caused immediate hemolysis of the patients' washed red blood cells when resuspended in such serum. The hemolysis was further increased by subsequent equilibration of the suspension with carbon dioxide. The addition to the serum of sodium bicarbonate in 10 or 20 millimolar concentration caused striking decrease in the hemolytic action of carbon dioxide. The addition to the serum of sodium chloride in 20 millimolar concentration did not inhibit hemolysis by carbon dioxide. Therefore, the degree of hemolysis was apparently related to change in pH. The hemolytic action of carbon dioxide was prevented by the addition to whole blood of sodium citrate (0.3 per cent), of potassium oxalate (0.2 per cent) and of potassium cyanide (0.13 per cent).

Washing the red blood cells of the three patients before resuspension in their own plasmas containing heparin or in their own serums did not modify the hemolytic action of carbon dioxide and of lactic acid, or the protective action of sodium bicarbonate. However, such washed red blood cells suspended in physiologic salt solution were not hemolyzed by carbon dioxide at a partial pressure of 40 mm. of mercury or less. Replacing the plasmas or serums of the patients with the plasmas or serums of normal subjects of all blood groups did not modify the hemolytic effect. On the other hand, resuspension of the washed red blood cells of normal subjects (Group O) in the plasmas or serums of the three patients did not result in hemolysis upon exposure to carbon dioxide or to lactic acid. Thus the essential peculiarity in these patients apparently resides in the red blood cells, whereas a factor essential for hemolysis is common to the plasmas or serums of the patients and of all normal subjects investigated.

The plasma or serum factor essential for hemolysis of the abnormal red cells by decreased pH was destroyed by heating for 30 minutes at 50 or 60°C., and was not resupplied or reactivated by the addition of complement in the form of 20 per cent of fresh human serum, of fresh guinea pig serum in a proportion of one part of a 10 per cent saline dilution to nine parts of human heated serum, or of a 0.4 per cent solution of methylene blue in a concentration of 10 or 100 mg. per 100 cc. of blood. Inhibitory factors were not demonstrable in heated serum, since the addition of such serum to an equal quantity of unheated serum reduced the amount of hemolysis no more than the addition of the same quantity of physiologic salt solution. Washed red blood cells of the patients, obtained from samples of whole blood containing sodium citrate, potassium oxalate or potassium cyanide, when resuspended in serum showed the hemolytic reactions of untreated cells. On the other hand, the washed red blood cells of the patients were not hemolyzed when suspended in serum containing the inhibitory substances listed above.

DISCUSSION

It is apparent that the red blood cells of these patients, both in vitro and in vivo, are abnormally susceptible to hemolysis in plasma of increased acidity within the physiologic range of pH variation. A thermolabile factor essential for this hemolysis was found in the patients' plasmas and serums and in the plasmas and serums of normal subjects. This hemolytic system differs from that of paroxysmal hemoglobinuria from cold (Donath-Landsteiner reaction[9]) in the following respects: (1) hemolysis occurred without previous chilling; (2) the essential abnormality resided in the red blood cells; (3) the plasmas showed no apparent abnormality or hemolytic action on normal cells; (4) the plasmas of normal subjects exhibited the same behavior as the plasmas from the patients; (5) since the addition of complement did not reactivate the heated serums, the hemolysis described above did not depend upon an antigen-amboceptor-complement system. Because the red blood cells of these patients showed a normal resistance to hemolysis by hypotonic salt solutions, the abnormality characteristic of congenital hemolytic jaundice was not present.

The oral administration of acid-forming salts clearly increased intravascular hemolysis. Although the oral administration of alkaline salts decreased intravascular hemolysis, withdrawal of alkali produced such striking and prolonged elevations of hemoglobinemia and hemoglobinuria that it is considered inadvisable as a therapeutic measure.

In one patient, previous splenectomy apparently had not altered the fundamental abnormality of the red blood cells or significantly decreased the anemia, but had eliminated the increased hemoglobinuria during sleep. It is probable that the slight decrease in the pH of the peripheral blood during sleep may not entirely reflect the acidity of certain regions of the body which results from the

relatively inactive circulation at such times. Destruction of red blood cells is thought to occur within the splenic pulp, where anatomic and physiologic observations[10] have demonstrated an ample opportunity for circulatory stasis to occur. Therefore, it appears likely that splenectomy in one patient had removed an organ which in the other two patients may provide a region with sluggish circulation and consequently with a pH materially lower during sleep than that of the general circulation.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23481

PRESENTATION OF CASE

A twenty-six-year-old, white, pregnant housewife entered the hospital with intermittent abdominal pain.

Her pregnancy was of six and a half months' duration and had been entirely uneventful. Twenty-four hours before entry she had slight pain in the epigastrium which became more marked and was accompanied by two attacks of vomiting.

She had had one normal pregnancy three and a half years before entry. Her past history and family history were otherwise negative.

Physical examination revealed a well-developed, well-nourished, pregnant woman having uterine contractions every two minutes and lasting from fifteen to twenty seconds. The contractions were very painful, and the abdomen was very tender. There was no show, and rectal examination revealed no dilatation of the cervix. Fetal heart sounds were heard. The blood pressure was 110 systolic, 70 diastolic.

The temperature was 98.6°F., the pulse 88. The respirations were 20.

She was given morphine and scopolamine and for the next twelve hours continued to have uterine contractions occurring every two to four minutes. Thirteen hours after admission the abdomen was soft, and no unusual tenderness could be made out. Shortly after that uterine contractions became almost continuous, and fifteen hours after admission a living premature child was delivered spontaneously followed by the spontaneous expulsion of intact membranes and placenta. The child died three and a half hours later. The patient was returned to her bed in excellent condition, and six hours later her temperature was 98.6°F., her pulse 88, and her respirations 20. A urine examination showed the slightest possible trace of albumin. Twenty-four hours later she had a chill with a temperature of 101°F., a pulse of 120 and a respiratory rate of 30. There was some distention of her abdomen, but no spasm or localized tenderness. For the next three days her condition remained essentially unchanged, with the temperature fluctuating between 99.5 and 101.5° F. The blood showed a red cell count of 4,340,000 with 70 per cent hemoglobin, and a white cell count of 16,500 with 85 per cent polymorphonuclears. Her abdomen remained somewhat distended, and on the eighth hospital day she was given a magnesium sulfate, glycerin and water enema. Almost immediately after the enema she had a severe chill; her temperature rose to 104°F., her pulse to 160 and her respirations to 30. The abdomen became very much distended, tympanitic, and tender throughout. Peristalsis was no longer audible. Two white cell counts made on that day were 3700 and 5400 with 77 per cent polymorphonuclears. The patient was placed on Wangensteen suction and an Ochsner regime. On the following day the white count was 16,900. Her physical signs persisted, and her temperature fluctuated between 104 and 106°F. On the eleventh day she was given a transfusion of 500 cc., and the following day she died.

DIFFERENTIAL DIAGNOSIS

DR. JOHN ROCK: This is the story of a twenty-six-year-old woman, six and a half months pregnant, with a negative past history, save for a previous pregnancy.

Her illness started with slight epigastric pain, which, however, became more marked during the twenty-four hours before entrance to the hospital. There were two attacks of vomiting. On admission there were uterine contractions of somewhat increased frequency and of short duration. The contractions were painful, and the abdomen was very tender. The systolic blood pressure was only 110, and the temperature, pulse and respirations were normal.

The pain in the epigastrium, which was slight at first, became more marked. Some complication of the pregnancy such as toxemia, might cause this, but there were no other signs or symptoms of toxemia; especially, there was no increase in systolic pressure. Premature separation of the placenta could cause it, but the course of labor and the normality of the product of conception rule this out. Rupture of the uterus gives pain, but also hemorrhage and shock. The course of labor and its results rule out a purely obstetric complication.

Non-obstetric causes of epigastric pain and vomiting severe enough to start a process which may eventuate in death are usually rather dramatically introduced. There is no indication here of gallstones, and rupture of a gastric or duodenal ulcer or rupture of the spleen would present shock or shock and hemorrhage. The pain from sudden rupture of a viscus is notoriously severe, not such as is described here. Pneumonia may give epigastric pain and vomiting, but is not to be suspected in this case.

Appendicitis usually starts with mild epigastric pain, and is occasionally associated with little or no rise in temperature. The pain usually recedes

to the right lower quadrant; but at six months the appendix is displaced upward some 5 cm., and the pain likewise. The character of onset and the location of symptoms strongly suggest appendicitis. In pregnancy this is more common, I think, in patients who have had previous attacks, but of course such a history is not necessary. It is said to be much commoner in the first two pregnancies than in later ones. This is the patient's second pregnancy.

Premature labor is induced by various causes, among them toxemia, which we have ruled out, and premature separation of the placenta. Intrauterine causes such as this, or placenta previa, hydramnios, multiple pregnancy, or death of the fetus, are also ruled out by the course and the result of labor. High fever, as a cause, is absent. Syphilis is the commonest cause, but would not account for the rest of the picture.

Appendicitis usually starts contractions. This description of the case suggests false labor for twenty-four hours, as there was no dilatation of the cervix, and then, true labor. I should make a diagnosis of appendicitis beginning before labor, and precipitating it. How far it progressed before delivery is hard to say — probably to abscess formation, though this is often not the case in pregnancy because of the displacement of the omentum. Rupture of an abscess, if it formed, would be expected with the sudden emptying of the uterus and the disturbance of anatomy. The relief of symptoms following delivery also suggests this time relation. The picture then became one of slowly progressing general peritonitis.

One might think of puerperal sepsis or peritonitis from a uterine source. The onset of definite signs of trouble as soon as twenty-four hours after delivery makes both diagnoses unlikely. The moderation of temperature range also argues against puerperal sepsis. All the physical findings strongly suggest a prompt peritonitis and very little resistance on the part of the patient.

I believe that the patient died of a ruptured appendix, giving general peritonitis. Appendicitis is a very difficult diagnosis to make in pregnancy anyway, and especially after it has precipitated contractions.

DR. HORACE K. SOWLES: How much of a factor do you think the enema was?

DR. ROCK: I think that the enema may have disturbed the adhesions or the setup in the peritoneum. I do not think it was an original cause.

DR. SOWLES: There was a very dramatic change at that point.

CLINICAL DIAGNOSES

Miscarriage.
Generalized peritonitis, ? etiology.

DR. ROCK'S DIAGNOSES

Ruptured appendix.
General peritonitis.

ANATOMIC DIAGNOSES

Acute appendicitis.
Appendiceal abscesses.
Peritonitis, acute purulent, generalized.
Involuting uterus, postpartum.
Thrombosis of the uterine veins, partial.

PATHOLOGICAL DISCUSSION

DR. TRACY B. MALLORY: The autopsy showed a general peritonitis as everyone had expected, and showed also two localized collections of pus in the immediate neighborhood of the cecum, one lying above and a little lateral to it, the other below and medial. Forming part of the wall of this second abscess cavity was the appendix, of which the medial third was blackish, contained a fecolith, and showed all the evidences of acute appendicitis. It had not perforated so far as we could make out grossly. The uterus and pelvic veins were entirely normal. There were thromboses in some of the uterine veins, but they were bland thrombi and showed microscopically no evidence of an inflammatory process. The degree of thrombosis was, I think, no more than might be expected a few days after delivery. I am quite sure that the source of the peritonitis was the appendix and that the uterus can be ruled out.

CASE 23482

PRESENTATION OF CASE

First Admission. A forty-one-year-old American housewife was first admitted complaining of swelling of the left side of the neck associated with a choking sensation.

The patient had been well until five months before entry, when she developed a persistent mucoid nasal discharge. The discharge was not associated with any other symptoms. Three months before entry she aborted a two-month fetus following the administration of drastic cathartics. Subsequently she bled profusely and entered a hospital, where she remained for four days and was discharged still feeling rather weak. Several weeks later, while stretching her arms upward, she suddenly felt a peculiar "catch" in the left side of her neck. This sensation persisted but occasioned no particular inconvenience. At the same time non-tender swelling of the left side of the neck was noted. The swelling gradually increased in size and was associated with a slight choking sensation in the throat and a feeling of fullness in the head. Two weeks before entry there was slight sore throat fol-

lowed by considerable hoarseness and dysphagia. These symptoms gradually subsided. On the morning of entry, while riding in a trolley car, she was suddenly seized with a spell of faintness, choking, extreme dyspnea and cyanosis which lasted for about ten minutes. Following this she felt quite well, although she was emotionally upset.

The patient had been married twenty years and had seven living children. The oldest was nineteen and the youngest two years of age. The only miscarriage was the one noted above. The patient weighed 220 lb. a year before admission and shortly before entry weighed 198 lb.

Physical examination showed a well-developed and obese middle-aged woman in no acute discomfort. The left side of the neck was swollen. The right pupil was larger than the left, although both were normal in configuration and reacted well to light and distance. The tonsils were symmetrically enlarged, red and boggy. In the right supraclavicular fossa and in the left anterior triangle of the neck were numerous olive- to plum-sized, firm, matted, non-tender lymph nodes. These were not adherent to the skin. The heart was normal. The lungs were clear. The abdomen was obese and pendulous but otherwise negative. Pelvic examination showed second-degree retroversion and old lacerations of the cervix.

The temperature, pulse and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 4,800,000, 70 per cent hemoglobin. The white cell count was 10,600, 70 per cent polymorphonuclears. The platelets appeared to be increased. Examination of the stools was negative. A blood Hinton test was negative.

X-ray examination of the chest showed that the diaphragm was normal in position and outline, and the costophrenic angles were clear. The hilar shadows were increased in size and density, and in the superoanterior mediastinum there was a shadow of increased density radiating from a point just above the aorta. Another film showed that the upper mediastinal mass displaced the trachea to the right and projected beyond the right side of the spine. The shadow was sharply outlined and there was a suggestion of lobulation of its borders.

On the day following entry a lymph node of the neck was biopsied. Pathological report was lymphoblastoma, giant follicular type. Subsequently the patient received intensive high voltage x-ray treatment, following which the nodes in the neck receded considerably, and the sensation of pressure was relieved. She was discharged on the sixteenth hospital day.

Second Admission (six years later). Following discharge from the hospital the patient remained in excellent general health until four years before entry, when she entered another hospital because of metrorrhagia. She remained in the hospital for two days and then returned home, although the metrorrhagia continued. Five months later she reentered the same hospital, and an ovarian cyst and the appendix were removed. Subsequently the metrorrhagia ceased. She remained well until about a year before entry to this hospital, when she noticed slight swelling of the nodes above the clavicle on the right side of her neck. Several months later she noted shortness of breath on exertion and increasing weakness. She re-entered another hospital five months before returning here because of pain in the left flank associated with urinary frequency and nocturia. Diagnoses of pyelitis and left ureteral calculus were made. She remained in the hospital for a short time and was discharged improved, although there was residual mild discomfort in the left flank. During the succeeding five months dyspnea increased in severity, and eventually orthopnea ensued. There were anorexia and a cough productive of small amounts of whitish sputum. For a month preceding re-entry she was confined to bed because of breathlessness. There had been loss of 20 lb. during the five-month period preceding entry.

Physical examination showed a well-developed and nourished woman sitting up in bed, mildly dyspneic. There was generalized lymphatic enlargement, the nodes being discrete, soft, nontender and measuring up to 1.5 cm. in diameter. The trachea was deviated to the right. There was dulness in the left upper chest anteriorly, merging into the cardiac dulness. In this region fremitus was diminished. In the left axilla at the level of the sixth rib there were bronchial breathing, pectoriloquy and a few transient crepitant rales. At the left base posteriorly, beginning at the angle of the scapula, there were dulness to flatness, diminished tactile fremitus, bronchial breathing and pectoriloquy. The heart was essentially negative. The blood pressure was 120 systolic, 80 diastolic in both arms. The abdomen was slightly distended, and there was shifting dulness in the flanks. The liver extended two and a half fingers beneath the costal margin, and the right kidney was displaced downward. The tip of the spleen extended two fingers beneath the costal margin, and there was a questionable soft mass in the left paraumbilical region. The remainder of the examination was negative.

The temperature was 100°F., the pulse 110. The respirations were 30.

Examination of the urine showed a specific gravity of 1.015; there was no albumin. The sediment contained many white blood cells but no

red blood cells or casts. The blood showed a red cell count of 4,500,000 with a hemoglobin of 70 per cent. The white cell count was 7250, 77 per cent polymorphonuclears. The stools were negative. The nonprotein nitrogen of the blood was 17 mg. and the serum protein 6.3 gm. per cent.

The chest showed a large, sharply defined mass in the upper mediastinum. The lower two thirds of the left lung field was homogeneously dull, and the dulness rose in the axillary line. The heart was slightly displaced to the right. An intravenous pyelogram showed a low left kidney and impaired function on the right side.

Intensive high-voltage x-ray therapy to the chest and abdomen was instituted, and the patient improved rapidly, her temperature returning to normal. On the twenty-first hospital day an x-ray film of the chest showed fluid completely obscuring the details in the right chest. The lower two thirds of the pleural cavity had a curved upper border and a thin layer of fluid extended to the apex. There was also a collection of fluid at the left base which obscured the outline of the heart. The mediastinum was not displaced, and there was no evidence of a mediastinal mass. The patient was discharged on the twenty-third hospital day.

Third Admission (two months later). Following discharge the patient became quite dyspneic, and two weeks later a left thoracentesis produced 1500 cc. of thick chylous fluid. Subsequently the dyspnea subsided but after three weeks recurred, and it was necessary to do a bilateral thoracentesis, at which time 1200 cc. of similar fluid was removed from both sides. The dyspnea was again relieved, but the patient began to have a dull aching pain in both costovertebral regions. The pain on the left side gradually became more severe and during the three days preceding re-entry was extremely distressing and accentuated by movement.

Physical examination at this time showed evidence of fluid in both pleural cavities, more on the left than on the right. The liver and spleen were still palpable, but no other masses were felt in the abdomen.

The temperature, pulse and respirations were normal.

The laboratory findings were unchanged.

X-ray examination of the chest showed fluid in both pleural cavities.

Subsequently, several chest taps were done and about 1000 cc. of chylous fluid was removed at each tap. Intensive x-ray treatment was given to the chest, and the patient was discharged improved on the eleventh hospital day.

Fourth Admission (two and a half months later). The patient remained well following discharge, but about ten days before re-entry again became dyspneic. This became progressively worse. There had been vague, generalized, intermittent abdominal pain.

Following oophorectomy three years previously there had been complete amenorrhea. For about a year preceding this entry, however, the patient had noted slight, bloody, vaginal discharge at irregular intervals. This continued off and on until three days before returning to the hospital, when she passed several small clots. There was no pain.

Physical examination was essentially unchanged. There was still evidence of considerable fluid in both pleural cavities. Pelvic examination showed an irregularly ulcerated, slightly enlarged cervix.

The patient received x-ray therapy, and another chest tap was done. The dyspnea and orthopnea continued but were slightly abated. A biopsy of the cervix showed early epidermoid carcinoma. Two other biopsies were reported as epidermoid carcinoma, Grade III. Radium was subsequently implanted within the cervix, and after its removal the patient was discharged, at the end of a month.

Final Admission (thirty-eight days later, six and a half years after her initial entry). Following discharge the patient remained well and was able to be up and about at home. There was no recurrence of dyspnea or vaginal bleeding. On the day of entry, while defecating, the patient had a sudden, profuse, vaginal hemorrhage, following which she felt weak and faint. A physician was called, and she was immediately sent to the hospital. There was no further bleeding until about fifteen minutes after entry, when she had another vaginal hemorrhage of about 400 cc.

A complete physical examination was not done because of her condition, but it was noted that she was pale and the lips were dry. There was no tenderness of the abdomen. The blood pressure was 70 systolic, 40 diastolic. The pulse was 140, and there was considerable fresh blood welling up from the vagina. Several clots were extracted, and a uterine pack inserted.

She was treated for shock, and seven hours after entry the blood pressure rose to 130 systolic, 90 diastolic. The pack was removed, and there was no further bleeding until twelve hours after entry, when about 1500 cc. of bright-red blood and clots were extruded. Her blood pressure was unobtainable, and despite the administration of supportive measures she died shortly afterward.

Differential Diagnosis

Dr. Marshall K. Bartlett: In this long and somewhat complicated case there are two separate and distinct neoplastic processes involved, each proved by biopsy and pathological examination. Neither lymphoblastoma nor carcinoma of the cervix is a rare condition in itself, but their association in the same patient is certainly unusual. More-

over, in this particular patient, each of these diseases presents certain unusual features which make the problem one of extraordinary interest.

Our first positive diagnosis is lymphoblastoma, giant follicular type. This is established on the first admission, and the patient's signs and symptoms are undoubtedly due to pressure of the nodular masses in the neck and superior mediastinum. The response to x-ray therapy was excellent, and she did not return to this hospital until six years later.

During this interval there are two events of interest which will bear mention. Two years after discharge from the hospital an ovarian cyst was removed in another hospital. This is of interest because, as we are told later in the history, it was followed by amenorrhea of three years' duration. The other point of interest is the left flank pain, associated with frequency and nocturia, which she experienced a few months before her second admission. Diagnoses of pyelitis and ureteral calculus were made. As will be seen later, recurrence of the lymphoblastoma is a more probable cause of this difficulty.

At the second admission the picture is that of a recurrent lymphoblastoma, with dyspnea and orthopnea. The most striking change is the presence of fluid in the abdomen and in both pleural cavities. Two weeks after discharge 1500 cc. of thick chylous fluid was removed from the left chest and soon after 1200 cc. was withdrawn from both sides. Numerous taps were subsequently done, with the character of the fluid remaining the same. Abdominal tap was apparently not done, but it seems fair to assume that the fluid in the peritoneal cavity would have been of the same type.

The presence of chylous fluid in the pleural cavity, non-traumatic in origin, is a rather rare condition of which only about 100 cases have been reported. The commonest cause is thoracic duct obstruction due to malignant disease, and this would seem to be the case here. This would also explain the fluid in the abdomen.

As might be expected, the response to x-ray treatment was less satisfactory at each successive entry, and except for the initial interval of six years, only a few weeks or months intervened between subsequent admissions.

At the first admission the cervix is reported to show old lacerations. No further note on this subject is made until the fourth admission, over six years later. At that time a history of intermittent vaginal bleeding for one year, following three years of amenorrhea, was obtained. Examination of the cervix showed it to be slightly enlarged and ulcerated and biopsy revealed epidermoid carcinoma, Grade III. Radium treatment was given, and the patient discharged from the hospital.

It does not seem probable that this lesion of the cervix could be a factor in producing the symptoms which we have attributed to pressure in the superior mediastinum, although the theoretical possibility of early metastasis from an unrecognized carcinoma of the cervix cannot be ruled out. It is more likely that the lesions in the chest are due to lymphoblastoma and that the only symptom referable to the cervix is the vaginal bleeding.

The final entry is brief and dramatic. The patient came into the hospital with a history of a profuse vaginal hemorrhage a few hours before admission. This massive bleeding recurred at intervals, and she died twelve hours after entry.

This constitutes another strikingly unusual feature of this case. Massive hemorrhage is a rare complication of carcinoma of the cervix. I have personally seen only one such case. In that instance it was the first symptom of the disease and occurred before any treatment was given. I have never seen massive hemorrhage from the cervix following radium and believe that it is an extremely rare complication.

Thus, we have in this patient lymphoblastoma with the unusual complication of thoracic duct obstruction and chylothorax and also carcinoma of the cervix with the rare complication of massive, and in this case fatal, hemorrhage following radium treatment.

Clinical Diagnoses

Lymphoblastoma.
Epidermoid carcinoma of the cervix.
Massive uterine hemorrhage.

Dr. Bartlett's Diagnoses

Lymphoblastoma with thoracic duct obstruction.
Carcinoma of cervix with massive hemorrhage.

Anatomic Diagnoses

Lymphoblastoma, generalized, giant follicular type.
Carcinoma of the cervix.
Necrosis of the cervix (radium implantation).
Pyometrium.
Chronic salpingitis.
Peritonitis, acute and chronic, focal.
Chylous hydrothorax, bilateral.
Pleuritis, chronic fibrous.
Mediastinal fibrosis and edema.
Pulmonary atelectasis.
Congenital atrophy, pelvic kidney.
X-ray dermatitis.
Peripheral edema.
(Massive uterine hemorrhage.)

Pathological Discussion

Dr. Tracy B. Mallory: This case, as Dr. Bartlett has pointed out, was in no sense a diagnostic problem, but was nevertheless of considerable interest. The primary disease was, as the biopsy proved, a form of so-called malignant lymphoma. In recent years pathologists have been attempting to classify with greater accuracy the group of diseases that were formerly included under this heading, and the giant follicular type is one of the more recent ones to be recognized. In it the lymphocytes tend to be arranged in readily recognizable follicles which closely simulate those seen in a hyperplastic node. In contrast to the hyperplastic node, however, these nodes tend to be made up almost exclusively of follicles; and the normal landmarks — the radial sinuses and the differentiation between cortex and medulla — are lost. The general experience has been that this type of case responds especially well to x-ray, at least on the first occasion that this form of treatment is used. The disappearance of the tumor masses is usually complete, and the interval before recurrence tends to be long. This case, with nearly six years' freedom from symptoms, is quite a typical one.

It is interesting to the pathologist, when successive biopsies are available or when we have a chance to check results by postmortem examination, to see whether the classification which we have originally made of the type of tumor holds good. In this case obvious large lymphomatous nodes were found in the retroperitoneal tissues, and they showed again the characteristic giant follicular picture. The mediastinum also was filled with diffusely infiltrating tumor, which had obliterated the thoracic duct and invaded the pericardium. In this area, perhaps because of the extensive radiation centered directly over it, recognizable tumor cells were very few and widely scattered, and the bulk of the tumor was made up of very dense and hyaline connective tissue; no traces of follicular structure could be made out.

The uterus showed extensive necrosis of the cervical segment and also of the endometrium. To the pathologist who looks at such a radiated cervix it is always surprising that events such as the fatal hemorrhage in this case are not much more frequent. The necrosis of the tissue is so extensive and deep that blood vessels are inevitably involved. Ordinarily, however, thrombosis must occur at least as fast as the vessels are eroded. Since a serious hemorrhage, such as occurred in this patient, is, as Dr. Bartlett stated, very rare, it is fortunate that it occurred in a patient who at best had only a few months more of life because of her associated condition. The new method of treatment for cervical carcinoma which Meigs and Dresser* have just outlined in a recent number of the *Annals of Surgery* should do a good deal to prevent this type of accident.

The only surprising features in this postmortem examination were the discovery that she still had two perfectly normal though atrophic ovaries and certain findings in the urinary tract. The left kidney, the kidney in which she had previously had some symptoms, was large, weighing 225 gm. At the time of autopsy there was no evidence of pyelitis and no stone. The right kidney was extremely small, weighing only 25 gm. It lay within the pelvis just below the lumbosacral joint. At its lower pole was a small fragment of functioning tissue, but the remainder was totally atrophic. Since the ureter also on this side was very narrow and distinctly short, it seemed most probable that this represented a congenital abnormality of the kidney rather than an acquired lesion.

*Meigs, J. V., and Dresser, R.: Carcinoma of the cervix treated by the roentgen ray and radium. Ann. Surg. 106:653, 1937.

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COMMUNICATIONS should be addressed to the NEW ENGLAND JOURNAL OF MEDICINE, 8 Fenway, Boston, Mass.

CO-OPERATION MUST CONTINUE

THIRTY years ago nearly 200 people out of every 100,000 of our population were dying from tuberculosis. Since then 3,500,000 men, women and children have died from this preventable disease but had the mortality rate prevailed an additional 2,500,000 would have perished. Today the tuberculosis mortality rate is 55 per 100,000 in this country, and there are more than 500,000 people sick with tuberculosis.

The chief concern of the thousands of men and women working with the national, state and local tuberculosis associations is to find tuberculosis in its early stage when cure may be more easily effected. The greatest number of deaths occur between the ages of fifteen and forty-five for which age group tuberculosis is still the leading cause of death. In spite of the steady improvement of diagnostic methods only 13 per cent of cases admitted to sanatoriums are found to be in the early stages of the disease, and this means there are far too many unrecognized cases in the community infecting their families and neighbors. Only by finding every single case can the disease be stamped out. Early examination, skillful diagnosis and prompt treatment are the factors which make the disease curable and preventable. Suspicious cases should be promptly examined, and examinations even where the disease is not suspected are important.

The recognition of tuberculosis in its incipiency is the greatest contribution that the practitioner can make in this campaign to eradicate the disease. In addition, as citizens, we should continue to contribute toward the financial support of the movement. Tuberculosis is everybody's problem. Appreciative as we are of the dangers, let us set a good example to the public in this world-wide fight against man's oldest disease enemy by buying Christmas Seals.

PNEUMONIA AS A CAUSE OF DEATH

SOME eighty odd years ago communicable diseases accounted annually for nearly 900 deaths per 100,000 population.* These diseases in order of their frequency as a cause of death were: tuberculosis, pneumonia, scarlet fever, typhoid fever, diphtheria, whooping cough, measles and smallpox. All but the first two have been practically eliminated as factors, and during the year 1936 they accounted altogether for less than 4 deaths per 100,000 population, whereas in 1856-1865 they were responsible for an average rate of over 330 deaths annually. This remarkable saving of life has followed the application of various discoveries in bacteriology, epidemiology and preventive medicine to the prevention and treatment of the diseases in question. Although tuberculosis in all its forms still produces an annual death rate of over 40 per 100,000 population, this represents a decrease of over 90 per cent from the 1856-1865 aver-

*Chadwick, H. D.: The Massachusetts Pneumonia Program. New Eng. J. Med. (in press).

age figure of 446. Pneumonia alone has successfully resisted efforts to lower it appreciably as a cause of death. For the decade 1856-1865 the average annual death rate was 107 per 100,000, whereas in 1936 it was 96, a decrease of only 10 per cent.

Our weapons for attacking the problem are limited. Probably the most important from the preventive point of view is the education of the public to view the so-called "common cold" with the proper amount of respect. For the protection of others, an individual so afflicted should be quarantined just as strictly as one with scarlet fever, and for his own good, he should be kept in bed for the first few days of his illness. Such procedures although idealistically advisable are practically impossible. The prophylactic inoculation of vaccines was recommended as long as twenty years ago and at present is being used experimentally among several large groups of young men, but proof of its effectiveness is too slim to warrant its adoption as a routine procedure.

So we are left with the facts that many thousands of cases of pneumonia are certain to occur in Massachusetts every year and that our only effective specific therapeutic agents are antipneumococcic serums. The Commonwealth through its Department of Public Health now furnishes type serums that are suitable for the treatment of more than half of all cases of pneumonia, and the Massachusetts Pneumonia Study has shown that the mortality rates of cases caused by these types of pneumococci can be reduced by approximately 50 per cent with their proper use. Furthermore, typing and serum distribution centers have been established throughout the State, and this service is available to all physicians. As previously mentioned, "proper use" is necessary; *adequate amounts of the correct serum must be administered early in the course of the disease.* More detailed information is given elsewhere in this issue of the *Journal* in a statement signed by the Pneumonia Committee of the Boston Health League. All physicians should take advantage of this opportunity of appreciably limiting pneumonia as a cause of death.

MASSACHUSETTS MEDICAL SOCIETY

A LETTER

To the Editor: At the October, 1937, meeting of the Council of the Massachusetts Medical Society, the Committee on Ethics and Discipline was instructed by the Council to proceed with appropriate action with reference to the letter of complaint in its possession in regard to Dr. N. F. De Cesare.

The Committee has followed these instructions with the result that I have received the following letter from Dr. N. F. De Cesare which I forward herewith to you for publication in the *Journal.*

November 22, 1937.

Channing Frothingham, M.D.,
President of the Massachusetts Medical Society,
8 Fenway,
Boston, Massachusetts.

Dear Doctor Frothingham:

I feel that I made an error in judgment in bringing suits against an officer and fellow of the Society and I am glad to apologize for so doing. I wish to take this opportunity to state sincerely that I had no intention of doing anything detrimental to the best interests and welfare of our Society in bringing these suits. I should like to make it clear to the members of the Society that the suits have been dropped.

You may publish this letter in the *New England Journal of Medicine* if you so desire.

With kindest regards, I remain

Sincerely yours,
N. F. De Cesare, M.D.

It is my sincere hope that with the publication of this letter the prolonged controversy in the Society originating over the matter referred to in this letter will be ended.

Channing Frothingham, M.D., *President,*
Massachusetts Medical Society.

8 Fenway,
Boston, Massachusetts.

SECTION OF OBSTETRICS AND GYNECOLOGY

M. Fletcher Eades, M.D., *Secretary,*
19 Bay State Road,
Boston

Case History No. 48. Premature Separation of the Placenta at Seven and a Half Months

Mrs. K. E., white, aged thirty years, was seen in the hospital at 5:30 a. m., November 22, 1935, in the thirtieth week of her first pregnancy. Her illness had started one hour previously when she

A series of selected case histories by members of the section will be published weekly.

Comments and questions by subscribers are solicited and will be discussed by members of the section.

arose from her bed to void her urine and passed bright red blood estimated as about 4 oz. There had been neither pain nor cramps, but bleeding had continued on the way to the hospital.

Her family history was negative. Her past history included scarlet fever, diphtheria, pneumonia, measles, mumps, pertussis and chickenpox in childhood, and numerous attacks of tonsillitis previous to tonsillectomy, which had been done at fourteen. Her catamenia started at seventeen, and were of the thirty-day type, lasting seven days. In 1933 she had been curetted for menorrhagia, for which no organic cause was found, and her periods had been normal since that time. Save for tonsillectomy and curettage she had undergone no operations.

She had been under observation for pregnancy since September 7, 1935, at which time she was in her twentieth week. Her physical examination at that time showed her to be a well-developed and nourished woman of 143 lb. Her color was good. Her breasts were normal, while her nipples, somewhat flat, showed the characteristic pigmentation and enlargement of Montgomery's glands associated with pregnancy. Heart and lungs were normal, and the abdomen was negative to palpation save for the presence of a uterus 11 cm. above the symphysis, corresponding to the dates of her pregnancy. The presentation and position of the fetus in utero could not be determined, and the fetal heart could not be elicited. Her measurements were 31 cm. interspinous, 29 cm. intercristal, 21 cm. external conjugate. Vaginal examination showed the perineum and cervix softened but intact, with ballottement in the anterior vaginal vault. The promontory was not reached. The outlet was slightly narrow with a bisischial measurement of 9.0 cm. Her blood pressure was 92 systolic, 60 diastolic, and a urine specimen was negative for albumin and sugar and had a negative sediment.

In the office visit which preceded her admission to the hospital her weight had increased to 154 lb., her blood pressure was 98 systolic, 60 diastolic, and her urine remained free of albumin. Quickening occurred in late September, and no symptoms of toxemia or bleeding occurred before November 22.

On admission to the hospital, she had a temperature of 98°F., a pulse of 78, and a blood pressure of 100 systolic, 70 diastolic. Examination of her uterus showed it to have increased in size since her initial visit to correspond to her dates. It was soft and elastic in consistence, but tender over the lower portion. The fetal heart was easily audible in the left lower quadrant of the abdomen, at the rate of 144 per minute.

Because of the continued bleeding, a diagnosis was made of premature separation of a normally implanted placenta, and immediate abdominal hysterotomy was advised. She was prepared at once for operation and anesthetized with 100 mg. of novocain intraspinally. The classical type of cesarian section was performed. The uterus was not mottled or infiltrated. The lower edge of the placenta, which had separated, was found beneath the hysterotomy wound. A male infant, weighing 3 lb., 5 oz., was extracted by the feet, breathed at once, and cried fairly vigorously after draining. On removal of the placenta and membranes some 250 cc. of fresh blood clot was sponged from the lower uterine segment. The remainder of the operation was uneventful, and the patient left the table in good condition.

Immediate recovery for the mother was excellent. The baby was clad at once in a premature jacket and placed in a heated Hess bed. Its lungs, however, never expanded fully, and despite inhalations of carbon dioxide and oxygen it became cyanotic, persisted in abdominal "sucking" respirations, and died of atelectasis five hours and forty-five minutes after delivery.

The mother's convalescence was normal.

Comment. In a rather summary discussion of this case it may be said that sudden painless bleeding in a woman thirty weeks pregnant at once suggests either placenta previa or premature separation of a normally implanted placenta. The latter diagnosis was chosen in this instance because of the tenderness over the uterus. Vaginal examination, however, would have been necessary to rule out absolutely the presence of placenta previa. On the other hand, the academic differential diagnosis is of minor importance in a woman not in labor with a living child in utero, who bleeds and continues to bleed at any time after the infant is deemed to be mature enough to have a reasonable chance of survival; in such a case abdominal section offers the child its best chance. The fact that this infant failed to survive and died of atelectasis does not alter the above principle.

Spinal anesthesia without preliminary medication with morphine or barbiturates was chosen in this case, as experience has shown that such preliminary medication and inhalation anesthesia, when administered to the mother, depress the respiratory center of the infant. Spinal anesthesia, moreover, was not contraindicated in the interest of the mother in this case as she was not in shock at the time of operation.

The debatable points in this case are these:

1. Would bleeding have ceased if the patient had been kept quiet under narcotics? The answer is *no,* because she showed no tendency to stop bleeding and was continuing to do so when treatment was instituted.

2. Would it have been better to control bleeding from below by the use of a vaginal pack and a tight abdominal binder? That would certainly have been the choice had the baby been dead in utero, had the patient been exsanguinated, or had she been twenty-six weeks instead of thirty weeks pregnant, as in each instance the infant would have had no chance of surviving. The question of the chances of survival of a living infant if delivered without trauma and without toxicity from medication, anesthesia, or both, must be answered in such a case on the spot. This infant, on statistical grounds, based on its birth weight, proved to have a 40 per cent chance of surviving as against a 60 per cent chance of succumbing. The fact that it failed to survive does not alter the fact that its interests were protected insofar as possible, albeit at some increased risk to the mother from the type of delivery chosen.

WHAT STRONG EMOTIONS DO TO US

Occasionally a person is said to be "beside himself" with anger. If not, perhaps he is "beside himself" with joy. The odd expression "beside himself" means not being his habitual self, but being quite different — so different as hardly to be recognizable. That is what strong emotions may do to us. They may transform us. Other common phrases give us further clues to the nature of emotional excitement. We speak of rage "seizing" us. We laugh or weep "without control." We feel "possessed" by fear. Or at a critical moment "something surges up within us." We are profoundly "moved," and then our actions seem no longer our own. The obscure and mysterious origin of emotional impulses and feelings has long fascinated students of human nature.

We find a hint regarding the source of emotional experience in an interesting fact — men and higher animals express emotions in similar ways. An angry man and an angry dog both show their teeth. The man mutters, the dog growls. When a man is stricken with terror he runs away; so does the dog. Or, if we are pleased, we laugh; the chimpanzee shows pleasure in the same manner.

Why is the expression of emotions in man and in higher animals so much alike? It can be explained by two facts. First, emotional expression, such as that for fear or joy or fright, is managed by lower parts of the brain. And, secondly, these lower parts belong to the primitive, ancient brain which is not very different in man and animals. The great distinction between man and animals is due to the higher parts of the brain. These parts are much larger and more complex in human beings than in the dog or the chimpanzee. For that reason the knowledge possessed by human beings can be much more complex, and their control of their surroundings can be much more complete. The activities in the higher parts of the brain are the basis of reasonable behavior. They are the basis of intelligence. They are associated with consciousness. Let the blood supply be shut off from these parts for only a moment and consciousness vanishes. We "faint away."

The activities in the lower parts of the brain display themselves outwardly in certain patterns. Examples are seen in the characteristic facial expressions and postures of joy, grief and anger. They have long been known and have been extensively studied. But, besides these surface signs of emotional excitement, there are profound bodily changes. Fortunately, just because emotional behavior is similar in animals and in man, we can study the effects of emotions in animals, and we can be fairly sure that facts thus learned will be true of man as well. Much of the new knowledge in this field has come from the natural and convenient hostility of the dog and the cat. The emotional excitement of each in the presence of the other is easily aroused, and the resultant effects have been examined. Let us consider these effects.

First, there is a stoppage of digestive activities. When a dog has been excited by a strange cat, the digestive fluids are no longer poured out. The salivary glands, for example, stop secreting saliva. We are ourselves familiar with the dry mouth of fear. Also, the churning actions of the stomach and intestines are abolished. Food may remain for hours in the stomach without being acted upon. This condition can be seen with x-rays in animals and also in human beings.

Again, there is a faster heart beat, and a change in the distribution of blood in the body. The large amount of blood needed in the stomach and intestines for digestion is sent away from those regions when digestion stops. This blood then flows in greater volume to the heart, to the brain and to active muscles.

Another change is an increase of sugar in the blood. If a cat is frightened by a dog for as short a time as three minutes, the sugar in the blood may promptly rise far above the normal level. There it may remain for some time. This change is as true for man as for the cat. Studies of Harvard football players in the highly exciting Harvard-Yale game revealed that their blood sugar was markedly increased.

Also there is poured out into the circulating blood a remarkable substance, adrenine, produced by small glands near the kidneys, called the adrenal glands. Adrenine has many effects in the body; prominent among them are the dilatation of the air tubes or bronchioles of the lungs and the abolition of the state of fatigue in muscles.

These various alterations which accompany excitement seem to be unrelated. We can see that they are related intimately with one another, however, if we consider the conditions under which strong emotions are aroused. In wild life or in the primitive conditions which surrounded our ancestors for untold myriads of generations, emotions were likely to be associated with prompt action. The emotion of rage, as we have noted, is associated with the impulse to attack, and the emotion of fear with the impulse to run away or escape. In the presence of a foe these emotions and the associated impulses are aroused. Whether one is the attacker or the attacked, the pursuer or the pursued, the struggle which follows may be a struggle for existence. The conflict is likely to be supreme. It may determine life or death. In these circumstances the great muscles of the limbs and the trunk may be used in intense and lasting effort. If we keep this central fact of struggle in mind, we have a key to the bodily changes which occur in great excitement. Let us try this key.

The digestive activities require a good circulation of the blood. When there is great effort, however, the blood is needed in the active muscles and in the nervous system which governs the muscles. Digestion is of secondary importance if life is at stake. The digestive process, therefore, can be temporarily stopped and the blood which it requires can be used elsewhere. The stoppage of the ac-

A "Green Lights to Health" broadcast given by Dr. Walter B. Cannon on Wednesday, November 17, and sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

tivities of the stomach and intestines and the shift of the blood from these digestive organs to the brain and to active muscles are thus seen to be appropriate.

The muscles carry on their labors most effectively if they are given sugar as a fuel. The increase of blood sugar, therefore, may be regarded as a preparatory supply of the material needed for vigorous muscular work.

Again, great effort is associated with a large increase in the amount of air that is breathed into the lungs and out again. It may increase tenfold as a consequence of struggle. The widening of the bronchioles by adrenine makes the movement of the air in and out much easier.

Furthermore, if activity continues and the muscles become fatigued, it is obvious that the action of adrenine, discharged at times of great excitement, is highly favorable to continued and efficient action of the muscles, for it abolishes the effects of fatigue and makes the muscles more responsive to nervous control.

Thus we see that these apparently disconnected changes are really parts of a single process. They can all be interpreted as directed toward making the body more effective in physical struggle. This mobilization of the bodily forces in great excitement accounts for the unusual power and endurance which have been noted in an emergency when a person has become excited and has had to put forth a supreme effort.

QUESTIONS

1. How do you account for the development of these violent changes in human beings under excitement?

Answer. These bodily changes may be regarded as the result of racial experience. They have been developed through innumerable generations of animals which have had to meet the demands of a fierce struggle for existence. For us, as we live in civilized society, the circumstances have been wholly altered. We have occasions for great excitement and for the attendant bodily changes, but relatively few occasions for the struggle that naturally follows. Thus, the student about to be examined may be intensely wrought up; the stockbroker watching a ticker may become as alarmed as if he suddenly faced a tiger in the jungle; and the patient waiting for an approaching operation may be profoundly worried and anxious. In these instances digestion may be upset, the heart may beat wildly, blood sugar may increase, and all the other changes which we have been considering may be brought about. But there is no consequent action. The body prepares for struggle when there is no struggle to engage in. The whole mechanism of reinforcing the body for great effort is set at work quite uselessly. The result is that this complex group of emotional reactions, valuable in more primitive conditions of existence, may become a real menace. Disorderly action of the heart, emotional dyspepsia or other harmful disturbances may result.

2. What can we do to avoid the bad effects of such emotions?

Answer. There are two ways of meeting such a critical situation. First, we may regard it reasonably and say to ourselves: "There is nothing to be done. Consequently we will not get excited and stirred up. Being excited is proper when there is something that requires action, but not now." This suggestion of a rational attitude may seem an impossible counsel of perfection, but experience has proved that it can be useful in preserving tranquillity. And the second way is to be followed when the first fails. If we are "seized" or "possessed" by powerful emotional impulses, we should let them go into action and should work them off in some sane and vigorous exercise.

3. But if outward circumstances which are upsetting us do not change, what should we do?

Answer. Various alternatives may be tried. First, of course, the occasion for it should be removed when that is possible. When that is impossible, at least three courses may be followed: experience with the emotion-provoking circumstance may be repeated until it loses its meaning and therefore has no further effect; it may be wholly avoided; or it may be minimized by finding other emotional outlets which substitute for it. Thus grief after the death of a loved one may be associated with a certain room: repeated use of the room, total avoidance of the room, or a deep change in internal feelings by earnest efforts to realize new values may be the means of meeting the distressing situation.

MISCELLANY

SPECIFIC TREATMENT FOR LOBAR PNEUMONIA

As the season of increased prevalence of lobar pneumonia approaches, it seems desirable again to call attention to certain matters pertaining to the disease, the importance of the recognition of types of pneumococcus infection and the application of specific treatment.

Lobar pneumonia is among the leading causes of death in Massachusetts. In 1936 there were 1944 deaths. It may be estimated that, in general, Type I and Type II pneumococci are together the cause of the disease in over one half of the cases. Of the higher types, Types V, VII and VIII pneumococci are the most common cause.

The case fatality rate of Type I pneumococcus pneumonia untreated with serum is approximately 25 per cent and of Type II, 41 per cent. The expected death rate in cases of pneumonia due to these two types can be reduced by early specific treatment. Of 1043 Type I cases in the Massachusetts pneumonia series which were treated with serum within the first four days of the illness, 145, or 13.9 per cent, died. Of 281 Type II cases similarly treated, 63, or 22.4 per cent, died. This experience in Massachusetts has demonstrated that specific treatment can be successfully used by physicians in general practice and may be expected to reduce the case fatality rate by about 50 per cent.

Success in specific treatment depends for the most part upon the early use of serum. Thus the type of pneumococcus infection should be determined at the earliest possible moment. The importance of the time element is emphasized in the experience in Massachusetts. Of 861 Type I cases treated during the first three days, 108 died (12.5 per cent) and of 182 treated on the fourth day, 37 died (20.3 per cent).

DETERMINATION OF TYPE OF PNEUMOCOCCUS INFECTION

The type of infection is more readily determined by examination of the sputum than by other means. The specimen should come from the lung with as little admixture of saliva as possible. It is desirable to obtain at least a teaspoonful of sputum, which is collected in a clean, wide-mouthed bottle or cardboard sputum box; this should be sent at once to the nearest laboratory equipped for typing. Special containers are available through local boards of health. *No antiseptic* should be added to the sputum; tuberculosis sputum outfits should not be used as they contain carbolic acid.

The Neufeld method of typing is rapid, simple and reliable. It is applicable to pneumococci from any source and often permits identification of type within a few minutes.

Typing will be done without charge at the State Bacteriological Laboratory, Room 527, State House, Boston. When necessary, typing of sputum from patients with pneumonia will be done during the night, week-ends, or on holidays. Such specimens should be left with the State House guard. All sputums showing Type I, Type II or Type V pneumococci, or any other type against which serum is available, are reported by telephone or telegraph, prepaid.

In addition to the State House laboratory, typing facilities are available elsewhere. The charges for the typing depend on the laboratory. Typing in Boston is done at the following hospitals: Boston City, Faulkner, Massachusetts Memorial, New England Deaconess and St. Elizabeth's. Elsewhere in the State facilities have been established for pneumococcus typing.

SERUM TREATMENT

At present in Massachusetts therapeutic antipneumococcic serums are available against Types I, II and V pneumococci. In the near future it is planned to distribute therapeutic antipneumococcic serums against Types VII and VIII. Antipneumococcic serums are available to physicians through the State Bacteriological Laboratory, Room 527, State House, Boston, or through approved laboratories, provided that:

(1) Sputum or other material from the patient is first typed and found to contain Type I, Type II or other type of pneumococcus against which serum is available.

(2) As soon as the patient is discharged, the physician agrees to make a report to the Massachusetts Department of Public Health on a form enclosed with the serum.

It is impossible to outline any schedule of dosage which will apply to every case. It is important to remember that successful serum therapy is chiefly dependent upon early and adequate specific treatment. Delay may entail the risk of death and the time-consuming administration of extra dosage of serum.

At present in Massachusetts, it is recommended, subject to the precautions which must be observed with respect to treatment with horse serum, that patients with Type I pneumococcus pneumonia be given an *initial dosage* of at least 60,000 units and that those with Type II or V be given at least 100,000 units. This initial dosage should be at least doubled, if

(1) Treatment is begun after the third day of the disease.

(2) The patient is over forty years of age.

(3) The patient is pregnant or in the first week of the puerperium.

(4) There is involvement of more than one lobe.

(5) Pneumococcus bacteriemia is known to be present.

Bacteriemia occurs in about one quarter of the cases with Type I and one third of those with Type II pneumococcus pneumonia and is a very serious condition. Thus, information in relation to bacteriemia is of great importance in treatment. It is desirable to make a blood culture in each case before the first dose of serum is given. If the blood culture is positive, or if negative and the progress of the case is unsatisfactory, it is desirable to continue to take blood cultures at intervals of about twenty-four hours or more often.

Blood-culture medium should be a part of the equipment of every physician who treats pneumonia. With the patient in a hospital and the laboratory nearby, culture medium in cotton-plugged flasks can be utilized. For use in the home, fluid culture medium in bottles, to avoid contamination in transit, will be available at such typing stations as are approved for blood-culture examinations. Blood cultures may also be sent for examination to the State Bacteriological Laboratory.

Additional individual doses of 40,000 to 60,000 units are recommended at intervals of every three or four hours in severe cases and in any case, if

(1) The temperature does not fall below 102°F. by rectum within eighteen hours of beginning serum treatment.

(2) Having fallen below 102°F. by rectum, it again rises above this level.

(3) Bacteriemia is demonstrated by blood culture regardless of the clinical course.

(4) There is evidence of a spreading lesion.

Failure to improve after the serum has been given *persistently* for seventy-two hours suggests the presence of some complication, such as empyema. A mistake in bacteriologic diagnosis is also possible. Another specimen of sputum should be sent to the laboratory for typing, blood culture taken and the patient examined for signs of complications.

Further information concerning the administration of serum will be found in the circular accompanying the serum. The directions given in the circular should be followed in detail.

NURSING SERVICE FOR PNEUMONIA

Clinical experience has shown that patients receiving prompt medical and nursing care have the best chances of recovery. In almost every part of the State, nursing service on a visit basis is available through the visiting nursing associations for patients not needing or who cannot afford a special nurse. In Boston the Community Health Association will give nursing care on a visit basis on the order of a physician. This association will be glad to co-operate with physicians by sending a specimen of sputum for typing.

PNEUMONIA COMMITTEE,
BOSTON HEALTH LEAGUE,
DR. FREDERICK T. LORD, *Chairman*,
MISS DOROTHY J. CARTER,
DR. HENRY D. CHADWICK,
DR. FRANK CRUICKSHANK,
DR. DWIGHT O'HARA,
DR. FRANK R. PHILBROOK,
DR. CONRAD WESSELHOEFT,
MISS MARGARET H. TRACY, *Secretary*.

THE TUBERCULIN TEST

"The tuberculin test should be routinely used by every general practitioner. This is the unqualified recommendation made by all outstanding tuberculosis specialists. The general use of the tuberculin test will help to diagnose the many cases of early symptomless tuberculosis that now escape discovery. It directs the attention of the physician to the hidden foci of infection that so often go unnoticed to the detriment of families and communities.

"Of the two accepted methods of giving the tuberculin test, the intracutaneous, intradermal (Mantoux) method is more accurate in that a known amount of tuberculin can be given and the dose increased if desired. For this reason, a slightly larger number of reactors can be found than is possible with the cutaneous (Pirquet) technic."

The above advice is quoted from *Tuberculosis Abstracts*

for December, 1937. The balance of the leaflet consists of plates incorporating the material used in an exhibit prepared by the National Tuberculosis Association for the meeting of the American Medical Association at Atlantic City in June, 1937. These show the simplicity of the tuberculin test and furnish graphic evidence of the advantage of P.P.D. (purified protein derivative) over O.T. (old tuberculin).

Any interested Massachusetts physician can secure, without cost, a copy of this issue of *Tuberculosis Abstracts* by addressing a postal card to the Massachusetts Tuberculosis League, 1148 Little Building, Boston.

RADIO BROADCASTS

The third group of weekly broadcasts sponsored by the American Medical Association and the National Broadcasting Company concern diet. These dramatized health messages are intended to furnish supplementary material for health teaching in junior and senior high schools and are broadcast every Wednesday from 2:00 to 2:30 p. m. over the Red Network. The dates and subjects are as follows:

December 8 — It Takes All Good Foods. A well-rounded diet and how to get it.

December 15 — Vitamins, Minerals and Common Sense. More about a balanced diet in special relation to minerals and vitamins.

December 22 — Dietary Fads. Facts *vs.* fallacies in relation to prevalent false notions on diet.

December 29 — Milk from Farm to Table. The production, transportation, pasteurization and home care of milk; its place in the diet; processed milks.

1938 AMERICAN MEDICAL ASSOCIATION ANNUAL SESSION

The Board of Trustees of the American Medical Association has appointed Dr. Howard Morrow of San Francisco as general chairman of the Local Committee on Arrangements. Among other appointments of local subcommittees, Dr. Morrow has appointed Dr. F. C. Warnshuis as chairman of the Committee on Hotels.

Fellows are requested to send in their requests for hotel accommodations to Dr. F. C. Warnshuis, Suite 2004, 450 Sutter Street, San Francisco, California, giving names of members in party, type of accommodations desired and times of arrival and departure.

Assignment of accommodations and their confirmation will be made for each reservation request. Do not write directly to any hotel as *all* reservations will be cleared through the hotel committee.

Banquet rooms and special dinner reservations must be made through the hotel committee. The same rule applies to special boards and allied organizational groups.

San Francisco affords first-class hotels capable of providing accommodations for 15,000 fellows and members of their families. However, early reservations are requested to avoid confusion and to ensure individual choice.

Those planning to visit San Diego, Los Angeles, Santa Barbara, Del Monte, Yosemite, or other California cities are urged to write in advance for hotel reservations in these cities. Following the American Medical Association Annual Session, the Rotarians, Kiwanians and Shriners hold their annual sessions in California. It is quite probable that many of the members of these organizations will visit points of interest before their conventions, thereby creating heavy demands on local hotels throughout the State.

RECENT DEATHS

GRINNELL — Francis Browne Grinnell, M.D., formerly a resident of Charles River, died in England on November 17, after a long illness. He was fifty years of age.

Dr. Grinnell was graduated from Harvard College in 1909 and from Harvard Medical School in 1913. He immediately became a member of the Department of Preventive Medicine and Hygiene of the latter institution and was then appointed bacteriologist at the Infants' Hospital. He joined the staff of the Department of Bacteriology, Harvard Medical School, in 1922 and, at the time of his retirement in 1937, held the rank of assistant professor of bacteriology and immunology.

He had made many contributions to bacteriologic literature, and his studies concerning the dissociation of the typhoid bacillus were largely responsible for the modern improvements in the preparation of typhoid vaccine. He was a member of the Red Cross Sanitary Commission to Serbia in 1915, served in the Medical Corps of the British Army from 1917 to 1919, and belonged to the Society of American Bacteriologists.

His widow, the former Winifred S. Dean, two sons and three daughters, and also three sons by an earlier marriage, survive him.

GREENLEAF — Colonel Henry Simpson Greenleaf, Medical Corps, United States Army (Retired), of 71 Griggs Road, Brookline, died November 8 at the Peter Bent Brigham Hospital, Boston. He was in his sixty-eighth year.

Born at a frontier post, Fort Lapwai, Idaho, he went from high school in Washington, D. C., to Johns Hopkins University from which he graduated in 1892. In 1895 he received his degree from the University of Pennsylvania School of Medicine. After practicing medicine for a year in California, he was commissioned in the United States Army, where he saw active service in the Spanish War, the Boxer Rebellion and the Philippine Insurrection. As a result of his service he received the Order of the Dragon. During the World War he organized the general hospital and was surgeon to the Officers' Training Camp at Fort Snelling, Minnesota, later serving as department surgeon of the Panama Canal Zone. Dr. Greenleaf served a lectureship in tropical medicine at the University of Cincinnati. He retired from active military service in 1925. From 1926 to 1932 Dr. Greenleaf was New England zone surgeon for the United States Fidelity and Guaranty Company and was with the Hartford Accident and Indemnity Company until shortly before his death.

Among his fellowships were those in the Massachusetts Medical Society, the American Medical Association, and the American College of Surgeons. He was also a member of the Cincinnati Literary Club, Harvard Travellers' Club, Episcopalian Club of Massachusetts, Massachusetts Society of the Order of the Founders and Patriots of America, the Massachusetts Society of Examining Physicians, and the Southern Minnesota Medical Society (Honorary).

His widow, two daughters, and a son, Dr. Henry M. Greenleaf, survive him.

WINSLOW — Frederick Winslow, M.D., of 275 Clarendon Street, Boston, died November 25. He was sixty-four years of age.

After being graduated from the Roxbury Latin School, Dr. Winslow entered Harvard University from which he graduated in 1895. He received his degree from Harvard Medical School in 1900 and then studied two years in Germany and Austria.

A fellow of the American Medical Association and the Massachusetts Medical Society, he was also a member of the Odd Volumes Club and the Harvard Varsity Club. Dr. Winslow was a trustee of the Boston Library Society.

His widow, a daughter and a son survive him.

RICH — CHARLES E. RICH, M.D., of 65 Broad Street, Lynn, died October 2 at his home. He was in his sixty-fourth year.

A native of Townell, Maine, he graduated from Freeport High School, after which he attended Greeley Institute and the University of Vermont. In 1902 he graduated from Baltimore Medical College and served internships at the Baltimore Hospital, Baltimore, and Union Hospital, Lynn. Since 1906 Dr. Rich had been practicing in Lynn.

Among his fellowships were those in the Massachusetts Medical Society and the American Medical Association. He was a member of the Masonic Order, Ionic Club of Swampscott, Lynn Chamber of Commerce, Lynn Y. M. C. A. and Salem Country Club.

His widow, a brother, and two sisters survive him.

CLEMENT — GEORGE W. CLEMENT, M.D., of Mammoth Road, Pelham, New Hampshire, died November 28. He was in his eighty-eighth year.

Born in Dracut, Massachusetts, he graduated from Bowdoin College in 1869 and from the Harvard Medical School in 1873. He later studied at Vienna and in several German hospitals. He practiced fifty years in Roxbury before his retirement seventeen years ago.

Dr. Clement was a fellow of the Massachusetts Medical Society and a member and former president of the Norfolk District Medical Society.

Two daughters and a son survive him.

REPORT OF MEETING

ACADEMY OF PHYSICAL MEDICINE

The fifteenth annual meeting of the Academy of Physical Medicine, held in Philadelphia, October 19 to 22, was an outstanding success. The program was an excellent presentation of scientific physical medicine.

Research studies were reported by Professor Hudson Hoagland and his associates, of Clark University and the Worcester State Hospital, Dr. S. Katzenelbogen, of the Henry Phipps Psychiatric Institute, Professor Robert S. Harris, of Massachusetts Institute of Technology, and Dr. Henry C. Bazett, of the University of Pennsylvania Medical School.

New apparatus devised by Drs. Charles W. McClure, Boston, Gustav Bucky, New York and Eugen Weissenburg, Vienna, were demonstrated.

Clinical demonstrations at the Jefferson Medical College Hospital conducted jointly by members of its staff and by those from the University of Pennsylvania Hospital and Temple University Hospital were a feature of the program.

Symposia, addresses and clinical papers on physical medicine in the various specialties in medicine and on fever therapy, short wave therapy, colonic therapy and electrosurgery occupied the three-day session.

Among the essayists were: Drs. Frederic J. Cotton, Frank R. Ober, William D. McFee, Herman A. Osgood, Henrich Brugsch and Joseph H. Pratt, of Boston; Fred H. Albee, William S. Bainbridge, Anthony C. Cipollaro, Mortimer N. Hyams, Robert R. M. McLaughlin, William B. Snow and James W. Wiltsie, of New York; Patricia H. Drant, Chevalier L. Jackson, Thomas J. Leichner, Josef B. Nylin, Jacques P. Guequierre, Robert P. Sturr, B. S. Troedsson, William H. Schmidt, William A. Swalm, and L. M. Morrison, of Philadelphia; Franklin P. Lowry, of Newton, Mass.; Frank H. Krusen, of Rochester, Minn.; Sir Robert Stanton Woods, of London; Isi Gunzburg, of Antwerp; and Martha Brünner-Ornstein and Franz Ornstein, of Vienna.

Dr. R. Tait McKenzie, of Philadelphia, was elected president; Dr. Rolland A. Case, of Cleveland, first vice-president; Dr. William H. Schmidt, of Philadelphia, second vice-president; Dr. Herman A. Osgood, of Boston, secretary-treasurer; Drs. Franklin P. Lowry, of Newton, Mass., and Byron S. Price, of New York, directors for three years.

The next annual session will be in Washington, D. C.

NOTICES

REMOVAL

GERALD BLAKE, M.D., has moved his office to 212 Beacon Street, Boston.

BOSTON CITY HOSPITAL

The monthly clinicopathological conference will be held at the hospital on Wednesday, December 8, at 12 o'clock noon, in the Pathological Amphitheater.

JOSEPH E. HALLISEY, M.D., *Secretary*
Medical Staff.

UNITED STATES CIVIL SERVICE COMMISSION EXAMINATIONS

ASSOCIATE BIOCHEMIST (SYPHILIS RESEARCH), $3200 A YEAR

A vacancy exists in the Syphilis Research Center, Johns Hopkins Hospital, Baltimore, Maryland. Applications must be on file with the U. S. Civil Service Commission, Washington, D. C., not later than December 28, 1937.

Applicants must have successfully completed a full four-year course leading to a bachelor's degree in a college or university of recognized standing with major study in chemistry or chemical engineering.

PRINCIPAL SPECIALIST IN MATERNAL AND CHILD HEALTH, $5600 A YEAR
SENIOR SPECIALIST IN MATERNAL AND CHILD HEALTH, $4600 A YEAR
SPECIALIST IN MATERNAL AND CHILD HEALTH, $3800 A YEAR
ASSOCIATE IN MATERNAL AND CHILD HEALTH, $3200 A YEAR

Applications for these positions must be on file with the U. S. Civil Service Commission, Washington, D. C., not later than December 28, 1937.

Applicants must have been graduated from a medical school of recognized standing with the degree of M.D., and have served one year of internship.

CARNEY HOSPITAL

The Out-Patient Department of the Carney Hospital will hold its monthly meeting on Tuesday, December 7, at 11:30 a. m.

Dr. Louis E. Phaneuf will speak on "Management of Vesicovaginal Fistula."

WILLIAM J. MACDONALD, M.D., *Secretary*.

MASSACHUSETTS PSYCHIATRIC SOCIETY

The next meeting of the Massachusetts Psychiatric Society will be held at the Boston Psychopathic Hospital on Friday, December 10, at 8:00 p. m. Dr. Arlie V. Bock will speak on "Psychiatric Trends as Seen by an Internist."

W. Franklin Wood, M.D., *Secretary.*

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance), Tuesday, December 7, at 8:15 p. m.

PROGRAM

Presentation of cases.

Thrombosis and the Use of Heparin. Dr. C. H. Best, professor of physiology, University of Toronto.

Dr. John Homans will preside. Medical students and physicians are cordially invited to attend.

Marshall N. Fulton, M.D., *Secretary.*

GREATER BOSTON MEDICAL SOCIETY MEETING

There will be a meeting of the Greater Boston Medical Society in the Beth Israel Hospital Auditorium on Tuesday, December 7, at 8:30 p. m.

Dr. George Baehr, clinical professor of medicine at Columbia University and attending physician to the Mt. Sinai Hospital, will speak on "Clinical Significance of the Pathological Alterations in Bright's Disease."

K. C. Rosen, M.D., *President,*
D. B. Stearns, M.D., *Secretary.*

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Monday, December 6

Monday, December 6

*4 p. m. Physicians and medical students are cordially invited to attend a clinic presented by the medical, surgical and orthopedic services of the Infants' and Children's hospitals, in the amphitheater of the Children's Hospital.

Tuesday, December 7

*9 a. m. - 10 a. m. Boston Dispensary. Diseases and Injuries of the Knee Joint. Dr. John D. Adams.

11:30 a. m. Carney Hospital meeting, Out-Patient Department.

*8:15 p. m. Harvard Medical Society. Peter Bent Brigham Hospital amphitheater (Shattuck Street entrance).

8:30 p. m. Greater Boston Medical Society. Auditorium of the Beth Israel Hospital.

Wednesday, December 8

*9 a. m. - 10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

12 m. Boston City Hospital, clinicopathological conference, Pathological amphitheater.

*12 m. Clinicopathological conference. Children's Hospital Amphitheater.

Thursday, December 9

*9 a. m. - 10 a. m. Boston Dispensary. Allergy Clinic: Presentation of cases. Dr. Joseph Kaplan.

Friday, December 10

*9 a. m. - 10 a. m. Boston Dispensary. Lymphoblastoma: A clinical pathological survey. Dr. Edward A. Gall.

8 p. m. Massachusetts Psychiatric Society. Boston Psychopathic Hospital.

Saturday, December 11

*9 a. m. - 10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.

December 2 — Faulkner Hospital Clinicopathological Conference, 5 p. m.

December 2 — Trudeau Society, 4:00 p. m., Massachusetts General Hospital.

December 7 — Harvard Medical Society. Page 932.

December 7 — Carney Hospital meeting, Out-Patient Department. Page 931.

December 7 — Lawrence Cancer Clinic. Page 849, issue of November 18.

December 7 — Greater Boston Medical Society. Page 932.

December 8 — Boston City Hospital, Clinicopathological conference. Page 931.

December 9 — Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, 8:30 p. m.

December 10 — Massachusetts Psychiatric Society. Page 932.

December 20 — Boston Medical History Club. 8:15 p. m. Boston Medical Library.

April 4-8, 1938 — The American College of Physicians. Page 41, issue of July 1.

October 17-21, 1938 — Clinical Congress of the American College of Surgeons, New York City.

District Medical Societies

BRISTOL SOUTH

May 5, 1938 — 5 p. m., New Bedford.

ESSEX SOUTH

January 5 — Danvers State Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

February 2 — Council Meeting, Boston.

February 9 — Essex Sanatorium, Middleton. Clinic at 5 p. m. Dinner at 7 p. m. Speaker: Dr. John B. Hawes, 2nd. Subject: Dust and Disease.

March 2 — Lynn Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

April 6 — Gloucester Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

May 5 — Censors meet at Salem Hospital, 3:30 p. m.

May 11 — Annual meeting, Salem Country Club, Peabody. Dinner at 7 p. m. Speaker and subject to be announced.

FRANKLIN

Meetings will be held at the Franklin County Hospital, Greenfield, at 11 a. m. the second Tuesdays of January, March and May.

HAMPDEN

Meetings will be held on the fourth Tuesday in January, April and July.

MIDDLESEX EAST

Meetings will be held at the Bear Hill Golf Club, Stoneham, at 12:15 p. m. on January 12, March 16, and May 11.

MIDDLESEX NORTH

Meetings will be held at the Vesper Country Club, Lowell, on January 26 and April 27.

NORFOLK DISTRICT

January 25 — Hotel Kenmore. 8:15 p. m. The Management of Uterine Prolapse. Dr. Louis E. Phaneuf.

February 23 — Hotel Kenmore. 8:15 p. m. Dermatitis Venenata Due to Cosmetics and Industrial Irritants. Dr. John G. Downing. Discussion by Dr. Francis P. McCarthy.

March 29 — Hotel Kenmore. 8:15 p. m. Subject to be announced but to be related to diseases of the kidney. Dr. Albert A. Hornor.

May, 1938 — Annual meeting.

The censors meet on the first Thursdays of May and November in each year.

PLYMOUTH

Meetings will be held at 11 a. m. on January 20, March 17, April 21, May 19 and July 21.

SUFFOLK

January 19 — Joint meeting with Boston Medical Library.

March 15 — Joint meeting with Boston Obstetrical Society.

WORCESTER

At the following meetings, except the annual meeting, dinner will be at 6:15, to be followed by business session and scientific program.

December 8 — St. Vincent Hospital, Worcester.

January 12 — Worcester City Hospital, Worcester.

February 9 — Worcester State Hospital, Worcester.

March 9 — Memorial Hospital, Worcester.

April 13 — Hahnemann Hospital, Worcester.

May 11 — Afternoon and evening, annual meeting. Place and schedule of program to be announced.